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CARE AND DISEASES OF TROUT

By H. S. DAVIS

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INTRODUCTION

During recent years there has been a continually increasing demand for more and larger trout for stocking purposes. A decade ago most of the trout were planted as advanced fry, or at least before they reached a length of 3 to 4 inches, and outside of the commercial hatcheries very few fingerlings were fed for more than two or three months. To-day the tendency is to hold the fish for much longer periods, and several States are already planting thousands of 6 to 12 inch trout annually.

This change in stocking policy is due primarily to the fact that in thickly populated sections, where the streams are fished intensively, it has been found that even moderately good fishing can be maintained only by liberal plantings of large fingerlings and legal-size fish. It has been estimated by Embody (1927) that in the streams of New York State 95 per cent of the advanced fry and 50 per cent of the fingerlings die before reaching legal size. If this be true, it is obvious that much better results can be attained by planting large fish, provided excessive losses in the hatchery and rearing pools can be avoided.

It is a comparatively simple matter to produce advanced fry in large numbers with little loss, but if fish are to be held over the summer the trout culturist is confronted with quite a different problem. Difficulties of various kinds, undreamed of by the early

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trout culturists, must be met and overcome if the fish are to be kept healthy and growing rapidly through the summer.

Unfortunately, there has been a tendency in some quarters to minimize the difficulties and to give the impression that trout can be reared to a length of 6 inches or more with very little trouble. It is very difficult to obtain accurate figures on the losses sustained by the average hatchery, but it is believed that under present conditions they are considerably greater than is usually realized. Owing to the careless methods of recording losses practiced at many hatcheries, their true extent is often not appreciated.

On the other hand, there is every reason to believe that such heavy losses are unnecessary and, to a considerable extent, can be easily prevented. There are well-authenticated instances of small lots of trout that have been carried through the first year with a total loss of less than 10 per cent from the time the eggs were taken. No doubt this represents an exceptional condition that can not be duplicated on a large scale, but there is no reason why we should not try to approach this record.

There is always some loss among the eggs, especially before they are "eyed," which in many cases reaches 20 to 30 per cent and in some instances may be even higher. But from the time the eggs are "eyed" until the young fish begin to feed there is usually very little loss. The final absorption of the yolk sac, which compels the advanced fry to seek its food from other sources, marks a critical period in the life of the young trout, which is sometimes attended with heavy losses. From this time until late summer or fall the mortality is often heavy, and it is during this period that there is the greatest opportunity to cut down losses through the adoption of better methods of caring for the fingerlings. Usually little difficulty is experienced in carrying the fish through the following winter if they are carefully graded according to size so as to allow no opportunity for cannibalism.

The various species of trout differ more or less in certain respects, and consequently they should not all be handled in precisely the same manner if best results are to be obtained. Since the writer has had more experience with brook trout (*Salvelinus fontinalis*), the present paper is based primarily on this species. In some respects brook trout are less adaptable to hatchery conditions than rainbow and brown trout, and it is probably for this reason that many hatcheries experience more difficulty with brook trout than with other species.

CARE OF FINGERLING TROUT

The conditions under which fingerling trout can be reared to best advantage, both from the economic and biological standpoints, is a problem worthy of more attention than it has received. Before discussing this question, however, it will be advisable to digress for a moment and consider the natural habitat of young trout during the first few months of their lives.

As is well known, brook trout normally spawn in small, swift streams, and the young remain near the spawning grounds or work their way up into even smaller streams during the first summer. These small brooks usually contain few pools of any size, and there

is a perceptible current almost everywhere. Even in the larger brooks, where there may be occasional quiet pools, one does not find the small fingerlings in such places; on the contrary, they are almost invariably in the shallow riffles, where there is a decided current and the water is well aerated. I have emphasized this point because it is self-evident that the more nearly we can simulate natural conditions in holding fingerlings the better will be our chances of success. This means that, if possible, the fish should be held where they can be furnished an abundant supply of well-aerated water, and, furthermore, the water should show a perceptible current. In other words, conditions similar to those that obtain in hatchery troughs should be provided in order to obtain the best results.

Many trout culturists hold the fingerlings during the first summer in the troughs in which they were hatched or provide larger troughs for the purpose. While good results are often obtained under these conditions, there are several serious objections to this practice.

Probably no better method than the standard hatchery trough can be devised to insure that all the fish will be provided with an adequate supply of fresh water. It provides an ideal means of hatching the eggs and holding the fry, but after the fish begin feeding other factors must be taken into consideration. In the first place, the rapidly growing fish constantly require additional space if they are to be kept in a vigorous condition. Consequently, if the fish are to be held for several months it will be necessary to have many times the number of troughs required to hold the eggs and fry. Troughs are expensive, and to provide a sufficient number to hold the fingerlings through the summer will greatly increase the overhead. In fact, if the fish are given sufficient room for best results, the cost of rearing them will be increased out of all proportion to the results attained. For this reason the troughs usually are overcrowded, with the result that the fish are stunted and not infrequently contract diseases, which cause a heavy mortality.

For the reasons given above I believe that raceways (fig. 1) provide a much more satisfactory means of holding fingerling trout than troughs. Of course, the raceway is only a development of the fundamental idea embodied in the hatchery trough but, considering the number of fish it will support, is much cheaper to construct and operate. It has the further advantage that, if properly constructed, it provides an environment closely simulating that of fingerlings in nature. This is especially true if the raceway has a sand or gravel bottom, as should be the case wherever practicable.

The size of the raceways may vary widely, dependent on local conditions, but in all cases they should be much longer than wide and supplied with sufficient water to insure a good circulation. Unless there is an exceptionally large flow of water it is believed that the raceways should be not over 3 to 5 feet wide with a maximum length of 50 to 75 feet. Only a very slight slope on the bottom is necessary, and the water in the lowest part should be not over 12 to 15 inches in depth. In such a raceway it will be found that the fish will be scattered quite uniformly along its length, and there will be little danger of the majority collecting at one end.

Many trout culturists use much deeper water than is advocated here, and where there is a sufficient volume flowing through the race-

way this may prove satisfactory; but it is believed that with a relatively small supply more fish can be held safely in shallow than in deep raceways. This is due to the fact that it requires a greater flow to produce a current in a deep raceway than in a shallow one. It is true that it may not be possible to hold quite as many fish per unit area of water surface, but, on the other hand, it requires less water to provide a satisfactory circulation. This being the case, by spreading the water over a greater area a larger number of fish can be reared with a given water supply.

After the fingerlings reach a length of 3 to 4 inches they may be held safely in small pools in which there is a less rapid circulation than in raceways. Smaller fingerlings are often held in pools successfully if given plenty of room, but our experience indicates that in most cases it will prove more economical to hold such fish in raceways and, if thought advisable, remove them to pools later in the season. A very good arrangement is to provide pools to which the surplus fish can be transferred when the raceways become overcrowded.

Of course, the type of pools or raceways to be employed will depend more or less on local conditions. Where there is danger of the water becoming overheated this fact must be taken into consideration and the pools so constructed that there will be no possibility of this occurring. In rapid, well-aerated streams brook trout can withstand temperatures as high as 75° F. for a short time without serious injury. Under similar conditions rainbow and brown trout can survive considerably higher temperatures. However, such high temperatures may produce disastrous results in hatchery waters. Where the fish are crowded together in small pools there is very likely to be a deficiency in dissolved oxygen when the water becomes warm, and under such circumstances high temperatures are much more injurious. Furthermore, hatchery fish are usually not as strong and vigorous as those living in a natural environment and consequently succumb more quickly to adverse factors.

Ordinarily, a temperature higher than 60° F. should not be allowed in rearing pools, and, where practicable, the water should be kept below that temperature. Trout appear to grow most rapidly at 55° to 60° F., and at higher temperatures their vitality is lowered and they are more liable to contract some disease.

Overheating of the water can often be prevented by partially shading the pools and raceways. In fact, this is advisable even where there is no danger of overheating, as it has been found that small fingerlings usually do better when given an opportunity to escape from the intense heat of the sun.

Where the soil is light and porous it is usually necessary to construct wooden or concrete sides for the raceways, but, unless absolutely necessary in order to retain the water, it is not believed that the bottom should be of concrete. A dirt bottom is to be preferred, although it is usually best to cover this with a layer of sand or gravel.

In addition to providing more natural conditions for young trout, raceways have a distinct advantage over troughs in that they require much less attention. Troughs must be cleaned at least once a day, but with raceways this is by no means true. In fact, even in heavily stocked raceways it is usually unnecessary to clean them oftener than

FIGURE 1.—Raceways for fingerling trout at the Pittsford (Vt.) experimental hatchery

FIGURE 2.—Rearing ponds at the Pittsford experimental hatchery

once or twice a week, and I have known of instances when they were allowed to go for several weeks without any attention other than to clean the screens so that the water could circulate freely, and this without any apparent injury to the fish. In an experiment conducted at the bureau's Pittsford (Vt.) experimental hatchery, fingerling brook trout were allowed to remain in a raceway from April to October without its being cleaned during that time, and there was virtually no mortality throughout the summer. Moreover, the fish were more vigorous, better colored, and made more rapid growth than those held in troughs. There were about 6,000 fingerlings in the raceway, which was 4 feet wide, 37 feet long, and had an average depth of 10 to 12 inches. The water supply fluctuated somewhat but averaged from 20 to 30 gallons per minute. This experiment is mentioned not with the intention of advocating that raceways should not be cleaned frequently, but simply to show that extreme cleanliness is unnecessary under such conditions.

As a matter of fact, raceways, like polluted streams, have a remarkable capacity for self-purification, which is accomplished in much the same way. The excrement accumulates on the bottom and supports a luxuriant growth of small organisms, including bacteria, algæ, Protozoa, and insect larvæ, which quickly causes its disintegration, so that the organic compounds are reduced to nontoxic, inorganic substances. The decaying excrement contains large numbers of chironomid larvæ, of which trout are very fond, and these, together with algæ, form a not inconsiderable portion of the food of the young fingerlings. It is not improbable that this "natural" food is largely responsible for the greater vigor of fish reared under such conditions.

There is considerable difference of opinion among trout culturists with respect to the age at which the fingerlings should be removed from the troughs. Personally, the writer is strongly inclined to the opinion that under ordinary circumstances the sooner the fish are removed to raceways after they begin to feed the better.

Probably the most common practice at the present time is to hold the fish in troughs for several months and then transfer them to raceways or pools in early summer. This practice appears to be predicated largely on the fact that while in the troughs the fish are under more immediate control and can be watched more carefully than when in raceways. While this is undoubtedly true, it is questionable if this outweighs the obvious advantages that fish in raceways enjoy.

It is held by those who believe in keeping the fish in troughs for the first few months that when they are transferred to raceways early in the season there is always considerable loss, which can not be accounted for by the number of dead fish recovered. Consequently, when the fish are finally removed their number is always much less than is indicated by the mortality record. This is undoubtedly true, but if the raceways are properly constructed and the fish graded frequently (as they should be) and protected from enemies, it is believed that this discrepancy will be not much greater than if the fish were held in troughs over the same period. In virtually all cases the so-called "uncounted mortality," which has been emphasized so much, is due either to improper construction of the raceways, so that many of the fish are able to escape; to the attacks of enemies, such as fish-eating birds; or to cannibalism. The first

two of these factors can be virtually eliminated by proper construction at comparatively small expense. It should be remembered that young fingerlings can wriggle their way through an almost inconceivably small opening, and all head and foot screens should be constructed accordingly. Protection from birds and other enemies can be secured by covering the raceways with wire netting.

In most cases it is probable that most of the "uncounted losses" are due to cannibalism, which is doubtless more rampant in raceways or pools than in troughs. It may seem paradoxical, but our experiments have shown that cannibalism is greatest among well-fed, rapidly growing fish rather than among those that are weak and underfed. Fortunately, cannibalism can be prevented by grading the fish at frequent intervals, so that only those of approximately equal size are held in the same compartment.

It is probably best in most cases not to transfer the fish to raceways until after they have learned to feed, although we have found in our experimental work that they can be taught to take food in raceways nearly as readily as in troughs. As previously pointed out, when the fingerlings reach a length of 3 to 4 inches they may be removed to pools that should be so designed as to simulate natural conditions as closely as possible. Pools with dirt bottoms are much superior to concrete, and unless the soil is of such a nature as to require wood or concrete sides these should also be dispensed with.

The size of rearing pools (fig. 2) will be largely dependent on local conditions, but in general it is believed that comparatively small pools are preferable. They should also be quite shallow, with a maximum depth of 3 to 4 feet, unless it is necessary to use a greater depth to prevent the water becoming too warm during hot weather. Needless to say, it is essential that the ponds have a copius supply of pure cold water, and they should be so constructed as to insure a good circulation throughout. The deepest part of the pond should be at the outlet, toward which the bottom should slope gradually from all sides, so that the pond can be easily drained and the fish removed with little trouble.

When the water supply is limited the ponds may be arranged in series, one below the other, which allows the same water to be used several times. Of course, this is objectionable from the sanitary standpoint, since a disease breaking out in the upper ponds would be transmitted immediately to fish in those below. For this reason it is not advisible to resort to such an arrangement unless it is made necessary by a limited water supply. However, with the exception of furunculosis, yearlings and older fish are seldom seriously affected by infectious diseases, and therefore the danger is not as great as would at first appear. It is well to bear in mind that, whenever ponds are arranged in series, fingerlings should never be placed in those receiving the drainage from ponds containing older fish. Yearlings and brood fish often harbor parasites, which, while not seriously affecting them, may produce disastrous results when transmitted to fingerlings.

CARE OF PONDS AND RACEWAYS

It is scarcely necessary to point out that raceways and rearing ponds should always be kept in a good, sanitary condition. No food

should be allowed to collect on the bottom, and the excrement should be removed frequently enough to prevent the water from becoming polluted. It is difficult to give detailed advice, since the care necessary to keep pools and raceways in sanitary condition will vary widely with conditions.

As previously pointed out, ponds and raceways do not require such close attention as troughs, because a certain amount of filth is taken care of automatically without detriment to the fish. The efficiency of self-purification naturally will vary with the number of fish contained in the pools. If the fish are relatively few, it may be necessary to clean the ponds only at infrequent intervals. The writer has known of instances when fish were kept in ponds with a dirt bottom which was cleaned only at intervals of several months. Although there was an average of one 6 to 8 inch fish per square foot of surface area, there was no evidence that the fish were affected unfavorably.

Under ordinary conditions, however, the accumulated filth should be removed more frequently to prevent any injurious effects from the decaying excrement. When cleaning the pools every precaution should be taken to avoid injury to the fish. The practice of going over the bottom with a broom or some similar implement, and thus stirring up the material that has accumulated, is highly objectionable, as it always results in considerable injury to the fish. Moreover, this practice necessarily removes most of the algæ, which is in itself inadvisable. Unless unduly abundant, algæ have a beneficial effect, and their growth should be encouraged. There are no more efficient agents in keeping the ponds in a sanitary condition than the algæ and associated organisms. Not only do they aid in aerating the water and keeping it free from objectionable substances, but it is found that trout feed on them to a considerable extent, and they, no doubt, perform much the same function in the trout's metabolism as do vegetables in the human diet.

A much better and more logical way to remove the excrement and other filth on the bottom of the pools and raceways is to employ some suction device that will avoid stirring up the material or injuring the fish. If this is not feasible, the lowest part of the pool should be connected with a drain, through which most of the filth can be drawn off with little trouble.

In this connection a word of caution should be addressed to those who are about to clean pools in which excrement and, more especially, surplus food has been allowed to accumulate for some time. Under such circumstances a layer of decomposing material may form on the bottom, which does not appreciably affect the overlying water as long as it is undisturbed. This layer contains noxious gases and other toxic substances, which are liberated as soon as the material is moved, and if the fish remain in the pool there is danger that they may be killed. In such cases the fish should be removed from the pond before any attempt is made to disturb the filth on the bottom. Even after the pond has been cleaned it is best to allow the water to flow through it for a short time before the fish are returned. When practicable, it is best to drain all ponds occasionally and allow them to remain dry for several days. If this is done once or twice a year, the ponds will be in better condition than if kept filled continuously.

TROUT FOODS

There is probably no question on which fish culturists differ more widely than in regard to what is the most satisfactory food for trout. During recent years this problem has received much attention, but we are far from reaching any general agreement as to the best and most economical diet for fish of various ages.

It is safe to say that, with the exception of the water supply, no single factor is of more importance in determining the success or failure of a hatchery than the daily diet of the fish. If we are to have strong, healthy fish they must be provided with suitable food, and it is no small problem to determine what food or what combination of foods may be relied upon to give the best results under average conditions.

In any consideration of trout foods it should be remembered that rapidity of growth is not the only factor to be considered. Too often in the past this has been virtually the only criterion used in evaluating a trout food. Rapid growth is very desirable, but it should not be procured at the expense of the health and vigor of the fish. The Federal and State hatcheries are raising fish to be liberated in natural waters, where they will be obliged to fend for themselves. It is very doubtful if the fat, lazy, pot-bellied fish so often seen in hatcheries are as well able to care for themselves, when thrown upon their own resources, as are fish that have not been pampered or subjected to an abnormal forcing process. For stocking purposes we need hardy, vigorous fish that can adapt themselves quickly to their new environment.

It is important to bear in mind that the trout culturist does not have an unlimited number of foods at his disposal, from which he can pick and choose. He is, in fact, bound hand and foot by considerations of cost and supply. Any food to be considered seriously must be obtainable at a reasonable cost, and there must be an adequate supply available at all times.

Available trout foods can be divided into three groups. In the first group are included fresh meats, such as horse meat and the liver, lungs, and spleen of cattle, sheep, and hogs. The second group embraces various dried products of animal origin, while in the third group we have the vegetable products, including wheat middlings, low grades of flour, shorts, soy-bean meal, Mexican pinto beans, etc. In a recent survey conducted by the Bureau of Fisheries it was found that sheep plucks are used to a greater extent than any other single food. Horse meat, which is used chiefly in the West, ranks next to sheep plucks. Other products that are fed in large quantities are beef liver, beef lungs, pig liver, cereal products, and fish.

As in the case of higher animals and man, fish require a certain amount of proteins, carbohydrates, and fats in their diet as well as various inorganic substances, such as lime and salt. These mineral constituents, of which there are a large number, are just as necessary as the proteins and fats. In addition, fish, like higher animals, require vitamins, which, although present in very minute quantities, are necessary for their well-being. It has not been determined definitely whether or not fish require all the vitamins that are essential to the health of man, but until it is proved otherwise it is certainly

advisable to assume that this is the case. A well-balanced diet should include these food constituents in the proper proportions to assure their being used efficiently and economically by trout.

Another factor that is essential to any trout food is its palatability. Obviously, if a food is to produce rapid growth it must be taken readily by the fish, and any attempt to force them to subsist on a food that is unpalatable will only result in failure. True, hunger may drive them to eat such foods in small quantities, but vigorous, rapidly growing fish can never be produced on such a diet. Our own experience has convinced us that this is the reason for the failure of some apparently good foods to yield satisfactory results. They may contain the proper proportions of proteins, carbohydrates, and fats as well as sufficient vitamins, but if they are not palatable the fish will suffer from partial starvation even though there is always a supply of the food available.

In any discussion of trout foods it is well to bear in mind that each species of trout presents a more or less independent problem, and that what may be true of one species does not necessarily apply to others. This is well illustrated by some recent experiments conducted by the Bureau of Fisheries, in which it was found that the addition of small amounts of cod-liver oil and yeast to the diet always proved beneficial in the case of rainbow trout, while with brook trout the results have just as consistently failed to show any benefit from the addition of these vitamin-rich products. Possibly the explanation of these diverse results may lie in the fact that rainbow trout appear to be more susceptible to vitamin deficiency than brook trout, and that the ordinary meat foods contain sufficient vitamins for the needs of the latter.

The following discussion of trout foods is based primarily on the requirements of brook trout, as this is the species used in most of the feeding experiments.

Although, as stated above, there is much difference of opinion among trout culturists as to the relative value of various food products, all agree that trout will not do so well for any length of time unless some fresh meat is included in the diet. This fact, which has been demonstrated time and time again, has recently led McCay and Dilley (1927) to postulate a hypothetical factor H, which is present in virtually all raw meats but is not found to any extent in cooked or dried products. The evidence for the occurrence of this hypothetical substance, which is easily destroyed by heat, is at present entirely indirect. Since there are other explanations that may be advanced to account for the failure of trout to flourish unless raw meat is included in the diet, it is believed that the presence of factor H should not be assumed without more conclusive evidence than has yet been forthcoming.

It has been the universal experience of fish-culturists that trout can be reared successfully on a straight meat diet, although there has been much difference of opinion as to which meat has given the best results. Everything considered, probably no food has given better results with young fingerlings than beef liver, and undoubtedly it would be used much more extensively were it not for the fact that its cost has more than doubled in the past two or three years. However, our own experiments with both brook and rainbow fingerlings

have shown that, in general, a mixed diet is preferable to one composed of a single meat product, and that better results are obtained when beef liver is mixed with beef heart than when it is used alone.

There can be little doubt that the results attained with various meat products are dependent on both their chemical and physical structure, and the latter should not be ignored by any means. This probably accounts for the fact that beef heart has been found to be superior to beef liver as a food for advanced fry and very young fingerlings. The heart can be ground into very fine particles, which are easily swallowed by the young fish, while the liver forms a thick, mushlike material not so readily separable into discrete particles. Furthermore, a considerable percentage of the ground liver is readily soluble in water, and there is therefore quite a loss in food value from this source.

As the fish increase in size they are naturally able to ingest larger particles, and the food is devoured more quickly. Consequently, there is less loss from solution, and liver will now produce a faster growth than heart, although there is usually a higher mortality. By combining heart and liver in a mixed diet we find that the growth is virtually as great as with liver alone, while the mortality is comparable to that of fish kept on a straight heart diet.

Pig liver and sheep liver, when fed straight to young fingerlings, has not given good results in our experiments. However, sheep liver produced a better growth with rainbow than with brook trout, although even with this species the results were not as satisfactory as with beef liver and beef heart. Both pig and sheep liver give better results when mixed with other products than when fed alone.

Horse meat, although used in large quantities by commercial growers in the West, has not been tested in carefully controlled experiments, so we have no means of comparing it directly with other trout foods. However, many growers report excellent results in feeding this product.

While only a few meats have been fed successfully to small fingerlings, a considerably greater variety is available for fish 3 to 4 inches long and upward. Fish of this size do much better on pig and sheep liver than very young fingerlings, and beef lungs and sheep plucks may also be fed successfully. Here again, however, a mixture of two or more kinds of meat usually gives better results than one alone.

Fresh fish of the coarser and cheaper grades have been utilized to a considerable extent for trout food, but in most cases the results have not been entirely satisfactory. It usually requires about twice as much fish as meat to produce an equal growth, and the trout are often not as healthy as those on a meat diet. Furthermore, it is much more difficult to keep fresh fish in good condition than meat, and several instances of trout having been killed by eating decayed fish have come to the writer's attention.

Owing to the marked increase in the price of most fresh meats during recent years it is believed that it is not economical to feed a straight diet of fresh meat to fish over 3 to 4 inches long, as fish of this size will thrive on a ration composed partially of dried products. Such a ration is much cheaper than one made up solely of fresh meat and in many cases will give virtually as good results and possibly even better.

The dried products that have given best results when substituted for part of the fresh meat in the diet are those of animal origin. Quite a variety of such products are now available, although, unfortunately, some of the best can be obtained only in limited quantities at present. Among the animal meals that have given sufficiently encouraging results in the bureau's experiments to justify their further consideration as trout food may be mentioned clam meal, shrimp meal, haddock meal (also called white fish meal), cod-liver meal, and dried skim milk. Probably there are other animal meals that should be included in this list but that have not been tested sufficiently in trout rations to justify an expression of opinion as to their probable value.

With the exception of clam meal and dried skim milk, the advisability of feeding these substitutes to small fingerlings is still somewhat questionable, and the writer would not advise using them on an extensive scale without a preliminary trial. Clam meal, which is composed of the dried refuse from clam canneries, has yielded by far the best results of any dried product used in our experiments. When mixed with beef liver and fed to small fingerlings the fish made a better growth than those fed beef liver and beef heart, which, with this exception, have given us the best results of any diet we have tried. The superiority of the clam-meal and beef-liver mixture was noticeable, both as regards growth and mortality; and the fish in this lot closely resembled wild fish in appearance, except that the coloration was not quite as brilliant.

Dried skim milk also produced excellent growth and low mortality when mixed with beef heart, and McCay and Dilley (1927) report equally good results with this product when combined with beef liver.

When combined with fresh meat, all of the substitutes mentioned above form a satisfactory food for larger trout, and the growth is often nearly, if not quite, as good as with a straight-meat diet. Moreover, there is no evidence that the health and vigor of the fish are impaired as a result of the use of these substitutes in the diet.

As previously pointed out, the cheaper meat products, such as pig liver, sheep liver, and sheep plucks, can be advantageously fed to large fingerlings and older fish instead of the more expensive beef liver and beef heart. These, when combined with animal meals, often give better results than when fed alone.

Below are shown the results of feeding experiments with yearling brook trout conducted at the experimental hatchery at Pittsford, Vt., for a period of 91 days. The dry meals were first mixed with water and then combined with ground meat in the proportions given.

Ration:	Per cent increase in weight, July 10 to Oct. 9
Pig liver 33⅓, clam meal 33⅓, shrimp meal 33⅓	209
Sheep plucks 50, clam meal 50	200
Sheep plucks 33⅓, clam meal 33⅓, shrimp meal 33⅓	190
Pig liver 50, clam meal 50	183
Pig liver	117
Sheep liver	112
Sheep plucks	51

A glance at the table will show that pig liver, sheep liver, and sheep plucks produced a much greater growth when mixed with clam meal and shrimp than when fed alone. Probably similar results could be obtained with haddock meal, cod-liver meal, or dried skim milk.

When using substitutes for meat, one of the most important problems is to determine the proportions of fresh meat and dry meals that will give the best results. Undoubtedly this will vary with the size and age of the fish and possibly with the species. Of course, from the standpoint of cost alone the smaller the percentage of fresh meat the better, but obviously there are limits to the extent to which meat may be replaced by a cheaper product; nor is it purely a matter of nutrition, since the physical consistency of the mixture must be taken into consideration. This factor has an important bearing on the readiness with which the food is eaten and also on the amount of nutriment lost in solution.

In preparing mixtures of fresh meat and meals it has been found best in most cases to first mix the dry meal with water to the consistency of a thick mush and then to incorporate it with the ground meat. When this mush is mixed with an equal quantity of fresh meat it forms a mixture of about the right consistency for feeding to small fingerlings. As most meals absorb from one and one-half to two times their weight of water, such a mixture contains about 15 to 20 per cent of the substitute, based on the dry weight. Our experiments indicate that this is about the maximum percentage of dried products that can be used successfully in the diet of young fingerlings.

Some trout culturists mix the dry meals directly with the ground meat, but it is believed that in most cases it is advisable to moisten the meal first. This is particularly true when a large percentage of the dried product is to be used. Owing to the ease with which dried skim milk dissolves in water it is necessary to add the dry milk directly to the meat, otherwise most of it will be lost.

It is well to bear in mind that, since fresh meats contain from 70 to 80 per cent of water, the addition of even a small percentage of dry meal directly to the meat greatly increases the amount of proteins, carbohydrates, and fats without a proportionate increase in bulk. In other words, such a mixture forms a more concentrated food than when the meals are moistened before mixing, and this fact should be taken into consideration in evaluating the mixture.

It is interesting to note that the animal meals that have been found to be of most value for trout are all rich in minerals and thus provide a class of food accessories in which meats are often deficient.

While it is not believed that it is advisable to use more than 50 per cent of moistened meals in the diet of young fingerlings, our experiments show that with older fish the percentage of fresh meat can be reduced considerably. As shown in the preceding table, a mixture of one-third meat with two-thirds clam and shrimp meal produced fully as rapid growth as when meat formed 50 per cent of the ration. Doubtless the percentage of meat could be reduced still further without affecting the fish adversely.

The vegetable products that have been used in trout foods include cereals and beans. Wheat middlings and low grades of flour are probably fed to a greater extent than other cereal products, although bran, shorts, and other meals also are utilized. These cereals are used to a much greater extent by commercial growers than by the State and Federal hatcheries. The reason for this is evident, as commercial growers are forced by stern necessity to use the cheapest food available with which they can obtain satisfactory results.

There is much difference of opinion as to the relative value of cereals and animal meals in the diet of trout and, unfortunately, we have but little experimental evidence bearing directly on the problem. Nevertheless, it is the writer's belief that the use of cereals in the diet of young fingerlings is inadvisable. This opinion is based on experimental evidence and also on the experience of years at the bureau's hatcheries. With such products as dried skim milk, shrimp meal, and haddock meal available at a reasonable cost it is not believed that it will prove economical in the long run to feed cereals to fingerlings. Such rapidly growing fish need relatively large quantities of food rich in proteins, which can be easily digested and assimilated.

In the case of older fish the evidence regarding the advisability of incorporating cereal products in the ration is not so clear. However, the fact that cereals are used to a much greater extent at commercial than at State and Federal hatcheries is in itself an indication that other foods are to be preferred where the comparative cost is not the deciding factor.

There is no doubt that trout can utilize cereals both for growth and maintenance, but to what extent they are able to do this has not been determined. While they are unable to digest raw starches, there is evidence that they can assimilate cooked starches to some extent, at least, as well as the proteins and mineral salts. When mixed with fresh meat cereals act as a "binder" and also absorb the meat juices, thus preventing waste by solution, and undoubtedly the results attained with cereals are partially due to this fact.

My own observations have led me to the conclusion that usually fish reared on a diet composed largely of cereals are not as strong and vigorous as those kept on a meat diet. There is a distinct tendency on the part of cereals to produce soft, fat fish, which may be satisfactory for market purposes, but it is doubtful if they are as suitable for stocking as trout that have been reared on a diet more nearly resembling their natural food.

There is also evidence that some species of trout may give better results with cereals than others. In our experiments rainbow trout have shown a distinct superiority over brook trout in this respect.

Experiments at the Pittsford (Vt.) hatchery with Mexican pinto beans, which are used to a considerable extent by western commercial hatcheries, gave satisfactory results with yearling rainbows, but with brook trout the growth was decidedly inferior to that produced by other substitutes. On the other hand, soy-bean meal gave much better results with brook trout than with rainbows. This meal is characterized by a high percentage of protein, which more closely resembles animal protein than other proteins from vegetable sources. It would, therefore, appear to be ideal for use in trout rations, but so far our experiments have failed to show that it is superior to some of the other vegetable products.

All cereals and beans should be cooked thoroughly before being mixed with meat. Cooking increases the digestibility and improves the consistency, so that these products can be fed to better advantage after cooking than before.

As previously mentioned, the use of cod-liver oil to supply vitamin deficiencies in the diet has given diverse results with brook trout and

rainbow trout. Experiments with rainbow fingerlings have shown that the addition of 1 to 2 per cent of cod-liver oil to the ration almost invariably produces beneficial results. With brook-trout fingerlings the results have just as consistently failed to show any benefit from the addition of the oil to the diet (Davis and James, 1924). Many trout culturists, however, make a practice of adding regularly a small percentage of cod-liver oil to the diet of both brook and rainbow trout and believe that the results are beneficial. At some hatcheries the oil is used only two or three times a week, but in larger amounts. It is claimed that in this way a greater laxative effect is obtained, which is considered a desirable feature. When trout are fed a well-balanced diet containing a considerable percentage of fresh meat it is believed that the use of dried yeast with the oil is unnecessary.

FEEDING METHODS

Meat and other foods should always be ground fine enough to be readily swallowed by the fish, but this is a matter that can be easily overdone. When the advanced fry begin feeding there is little danger of getting the food too fine, and it should be forced through the finest plate of the grinder several times in succession. As the fish increase in size they are able, of course, to swallow larger particles, and, if the food is not ground so fine they will take it more readily and less will be lost in the surrounding water.

In feeding small fingerlings most fish-culturists add a small amount of water to the food, so that the particles will separate readily in the water. However, some insist that it is better to place small portions of food at intervals on the bottom of the troughs, where the fish can break it up at their leisure. If carefully done, there is probably less loss by the latter method, but it is questionable if all the fish have an equal opportunity to obtain food.

When the fish begin feeding it is customary to feed at least five to six times a day, but as they grow older the number of feedings may be decreased gradually, and two meals a day are sufficient for the larger fingerlings. Older fish usually are fed once a day, although when a very rapid growth is desired it is probably better to feed both in the morning and at night. Of course, when the fish are fed twice a day it is not advisable to feed as much at one time as when they receive only one meal in 24 hours. Needless to say, the food should always be distributed over a considerable area, so that the fish will all have an equal opportunity to get their share. In feeding fish of any age it is always a good rule to give them only as much food as they will eat readily. It is very poor practice to allow food to accumulate on the bottom, because not only is such food a total loss but it will soon begin to decay and cause trouble.

Experiments at the Pittsford hatchery indicate that there is virtually no danger of overfeeding brook fingerlings under ordinary circumstances. In fact, it was found that the mortality was greater among fish that were slightly underfed than among those that were given all they would eat at each meal. There is evidence, however, that this does not necessarily apply to yearlings and older fish and that there is actual danger of overfeeding fish of this age, especially if the temperature of the water is relatively high and the fish slug-

gish. There is also probably a difference in different species in this regard. As every trout-culturist knows, rainbow trout are usually much heavier feeders than brook trout, and consequently there is more danger of overfeeding.

When trout are to be handled or transported any distance it is always advisable not to feed them for some hours previously. This serves a twofold purpose: There will be less excrement to accumulate in the cans and cause trouble, and the fish will stand handling better than if they had been fed recently.

IMPROVEMENT OF STOCK

One of the most important phases of trout culture and one that, remarkably enough, has received relatively little attention, is the necessity for improvement of the brood stock if we are to get the best results. Everyone recognizes that continuous and rigid selection is necessary in the case of domesticated animals and plants, but few seem to have realized the importance of this principle in trout culture. Indeed, the tendency in many cases has been in the opposite direction, for some of our commercial growers have marketed their best and largest fish and saved the remainder for breeding purposes.

On the other hand, some trout culturists have taken pride in building up a superior stock of brood fish, but usually the selection has not been carried on systematically for a term of years, as must be the case if permanent results are to be obtained.

Embody and Hayford (1925) have published a preliminary report on some breeding experiments with brook trout carried on at the New Jersey State hatchery. These experiments were conducted for the purpose of increasing the resistance of the fingerlings to disease and also to increase the rate of growth. By rigid selection they were able, in three generations, to produce an apparent increase in the resistance to disease and also marked improvement in the rate of growth. As a result of selection the average size of the fingerlings on July 1 jumped from 2 to 4 inches in length. This is certainly a remarkable result for only three generations of selective breeding and indicates what may be accomplished if the experiments are continued for a term of years.

The Bureau of Fisheries has recently embarked on an extensive program of selective breeding at its experimental hatchery at Pittsford, Vt. This work is to be carried on by mating individual fish rather than by mass selection as practiced by Embody and Hayford. The experiment has not been under way long enough to have yielded definite results, but there is no reason to doubt that it will be possible in this way to build up strains of fish much superior to the ordinary hatchery stock.

It has been found already that the young from a single pair are usually much more uniform in size than fish of mixed parentage, and that the offspring of different pairs show marked differences in the rate of growth under practically identical conditions. This was true even though the parent fish might differ but little in size or other characters. It is also notable that the young of certain parents showed much smaller losses than the young from other pairs that were apparently equally vigorous.

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There is considerable evidence to show that, in general, much better results can be obtained when a hatchery produces its own eggs than when the eggs are obtained from outside sources. This appears to be due to differences in the environment and indicates that eggs produced under certain conditions will develop much better and produce more vigorous young under the same conditions than when transferred to a different environment. Conditions at trout hatcheries vary widely as regards temperature of the water, dissolved minerals and gases, and other factors. In spite of such variations, experience has shown that most of them provide favorable conditions for trout, and it would be impossible in many instances to say which conditions are the best for trout culture. Undoubtedly, trout can flourish under a wide range of environmental conditions, but it does not follow that fish that have become acclimatized to one set of conditions can withstand an abrupt change to a quite different environment without detrimental effect.

This has been very noticeable at the Pittsford experimental hatchery, where brook-trout eggs from several sources have been hatched in adjoining troughs and the young reared under virtually identical conditions. In nearly every instance the local fish have suffered much smaller losses from various diseases than those hatched from eggs produced elsewhere. There have also been noticeable differences in this respect among fish hatched from eggs from different outside sources. There is no reason to believe that the diseases in question were brought in with the eggs, as they have been prevalent at this hatchery for years. The most logical explanation is that the vigor and vitality of the fish were affected by the changed conditions.

In view of these facts it is evident that for the best results the trout culturist should produce his own eggs from carefully selected stock. Of course, it is recognized that in many instances this may be impracticable for one reason or another. In such cases it will probably be found that eggs from certain sources usually do better than those from others, and the trout culturist can govern himself accordingly.

PARASITES AND DISEASES

The parasites and diseases of trout constitute one of the most important problems with which the fish culturist has to deal. Wild trout are only rarely seriously affected by disease caused by the presence of parasitic organisms. No doubt this is due primarily to the conditions under which they live in nature. The swift, cold waters of the typical trout stream are about as poorly adapted to the development and diffusion of trout parasites as could be imagined. When wild trout are injured by parasites and infectious diseases it is ordinarily fish living in ponds or lakes, where conditions are more conducive to the spread of parasites, that are affected.

When trout, or other fish for that matter, are herded together in hatcheries, conditions are reversed, and exceptionally favorable opportunities are afforded for the development and spread of the numerous parasites and diseases to which trout are susceptible. Of course, this is simply another application of the well-known principle that in the domestication of animals and plants we are, to a certain extent, running counter to natural laws, with the penalty of eternal vigilance, which this entails.

There is no reason to believe that the various parasites and diseases that are found at hatcheries have developed as a result of the domestication of trout. Undoubtedly, they all occur to some extent in wild fish, to which they ordinarily cause little or no injury; but when the fish are crowded together in hatchery troughs or pools there is every opportunity for the rapid increase and spread of parasitic organisms, resulting in the outbreak of epidemics with consequent heavy losses.

The control of these parasites and diseases is a problem of the greatest importance, and no trout culturist can hope to cope with them successfully unless he is familiar with the more essential facts. In the following pages no attempt has been made to deal with the subject in a technical or exhaustive manner. On the contrary, the sole object has been to give the essentials regarding each disease in as nontechnical terms as possible, so that the fish-culturist may be prepared to deal with it intelligently should the necessity arise, as, no doubt, will frequently be the case.

The great majority of the infectious diseases of trout are caused either by bacteria or Protozoa. As is well known, the bacteria are very minute organisms usually classified as plants, although they possess many characteristics that differ widely from those ordinarily associated with plants in the popular mind. On the other hand, the Protozoa are regarded as animals; but here, again, we must note that they are very different from the popular conception of an animal. The Protozoa are all very small, although much larger and more highly organized than the bacteria. Some of the larger Protozoa are visible to the naked eye, but the great majority are strictly microscopic, and some are visible only under a comparatively high magnification. The only characteristic that the Protozoa can be said to possess in common is that they are all composed of only a single cell, although even here, as in the case of the Myxosporidia, which are characteristic fish parasites, the distinction sometimes breaks down. Of more importance from the practical standpoint is the fact that the Protozoa are usually very delicate organisms easily killed by drying or by chemical agents. Many of them have a very complicated life history, a knowledge of which is essential to the devising of effective methods of control.

In addition to bacteria and Protozoa, the parasites of trout include several species of worms; but these are usually not as injurious as the former. In fact, Gyrodactylus is the only parasitic worm that ordinarily has to be considered by the trout culturist. Belonging to quite a different group is the parasitic copepod Salmincola, which is a member of the great group of Crustacea, a group that includes the "water fleas," crabs, crayfish, and shrimp.

Among the plants the only parasites, aside from bacteria, that affect trout are the water fungi, or Saprolegnia, which may attack fish under certain conditions. As fungus is always easily recognized, infections by this parasite have assumed an importance among fish-culturists out of all proportion to its actual potentialities for harm. Usually the appearance of fungus is an indication of the presence of some less conspicuous and more insidious agent, which is the real cause of the trouble. In other words, the appearance of fungus is a warning signal that no fish-culturist should disregard, but which in itself is relatively unimportant.

It not infrequently happens that fish may be affected at one time with two or more entirely distinct diseases. This, of course, complicates the situation and results not only in a much higher mortality than would otherwise be the case, but also greatly increases the difficulties of control.

From the practical standpoint it is of the greatest importance whether any particular parasite is external or internal. If it is an external parasite—that is, lives on the body, fins, or gills of the host—it is possible in most cases to apply some chemical that will destroy the parasite without serious injury to the host. Many substances have been employed for this purpose, the most widely used being a solution of common salt (sodium chloride). This is a very effective treatment for such delicate organisms as Protozoa and fungi but is ineffective when dealing with bacteria and such resistant animals as worms and copepods. In hatchery practice the salt is usually simply distributed throughout the trough, the water supply having previously been cut off. When the fish begin to show signs of distress and turn on their back or side the water is again turned on and the fish quickly recover. As it is impossible to control accurately the strength of the solution under such conditions, a better method in some instances is to dip the fish for a short time in a 3 per cent solution. This method requires less salt and the treatment can be controlled more readily.

Other chemicals extensively used for killing external parasites are copper sulphate and potassium permanganate. These chemicals are especially indicated when dealing with bacterial infections but are also effective against Protozoa. They are sometimes used in a very weak solution (1 part of the chemical to 50,000 to 100,000 parts of water), in which it is necessary to allow the fish to remain some time in order to kill the parasites. Weak as they are, these solutions will also kill the fish if they are allowed to remain immersed too long.

Owing to the labor involved and the danger of seriously injuring fish by leaving them for too long a time in a weak solution, the writer believes that in most instances it is preferable to use a much stronger solution, in which the fish are immersed for a very short time. Since the fish must be closely watched during the process, it is felt that there is less danger of their being badly injured than when a weaker solution is used, and the treatment is certainly as efficient, if not more so, in its effect on bacteria or other parasites.

In treating trout with copper sulphate, a solution of 1 part copper sulphate (by weight) is dissolved in 2,000 parts of water. The fish should be dipped in the solution for one or two minutes and then transferred at once to running water. They will at first show very evident signs of distress but in most cases will recover fully in a short time. Even small fingerlings, unless they have been weakened previously by disease, are not permanently injured by the treatment if it is carried out properly. If the disease is well established, many of the fish may be so weakened as to be killed by the treatment, but such fish would undoubtedly have died in any event. Healthy, vigorous trout will survive immersion in a 1 to 2,000 solution of copper sulphate for several minutes.

If a galvanized vessel is to be used for the solution, it should first be painted on the inside with asphaltum or some similar substance

to prevent any chemical action between the copper sulphate and the walls of the vessel. It is more convenient to use the pulverized form of copper sulphate, which dissolves very rapidly, so that the solution is immediately ready for use. To guard against deterioration it is always advisable to make up a fresh solution immediately before it is to be used. The solution rapidly becomes weakened with use and consequently should be renewed frequently.

Probably the most convenient method of treating the fish is to handle them in a dip net, which can be lowered into the solution, taking care that the fish do not escape from the net. By this method it is possible to treat a large number of fish in a comparatively short time.

The control of diseases caused by internal parasites presents quite a different problem, because in most cases it is impracticable to attempt the use of medicines. Good results sometimes follow a change in the diet or the addition of some such substance as cod-liver oil to the food, but in general the successful control of such diseases must depend almost entirely upon prophylactic measures. It is most important to keep the fish in as healthy and vigorous condition as possible by providing suitable quarters with an abundant supply of cold, well-aerated water. Overcrowding should be avoided, and, of course, the greatest care should be exercised to provide a suitable diet. Every precaution should be taken to guard against infection from any source, and any implements or vessels that might possibly carry infection should never be used with healthy fish unless they have been thoroughly sterilized. It is especially important in the case of fingerlings that the water supply should not have come in contact previously with older fish. *Under no circumstances should fish of any kind be allowed in the spring from which a hatchery obtains its supply of water.*

Of course, the precautionary measures just mentioned are fully as important in the case of external parasites as in combating diseases caused by internal parasites, but they are emphasized here because they are virtually the only measures we have of dealing with diseases of the latter type. In the treatment of diseases the trout culturist must exercise eternal vigilance and be quick to recognize the first indication of an outbreak. Half the battle lies in the ability to diagnose a disease in its early stages and adopt appropriate measures for its control before it is too late.

For the sterilization of ponds and raceways, where large quantities of a disinfectant are required, either "bleaching powder," also known as chloride of lime, or freshly slaked lime (calcium hydroxide) should be used. Owing to its cheapness, the latter is ordinarily employed for the purpose and when properly applied is as effective as the "bleaching powder." The unslaked lime or "quicklime" may be used if preferred, but if the lime is first slaked by the addition of water it can be distributed much more evenly over the bottom of the pond. It can be applied in the form of a powder or as "milk of lime" (1 part of slaked lime to 4 parts of water). It should be kept in mind that calcium hydroxide unites with carbon dioxide in the air or water to form calcium carbonate, which has no antiseptic value, and for that reason it should be freshly prepared shortly before it is to be used.

Needless to say, solutions of slaked lime or of "bleaching powder" should never be allowed to come in contact with fish, as they are quickly fatal. On exposure to the air for a time the lime becomes converted into the carbonate, and in this form it is no longer injurious to fish. When practicable, a pond, after having been sterilized, should be allowed to dry out and be exposed to the sun for several days or, better, weeks. If it is not possible to do this, water should be allowed to flow through the pond for some time before it is used again for fish.

EXTERNAL ANIMAL PARASITES

GYRODACTYLUS

There is probably no external animal parasite that is more common and causes more injury to trout than the small trematode worms belonging to the genus Gyrodactylus. These worms occur at virtually all hatcheries, although only occasionally do they become sufficiently abundant to cause serious injury. It is probable that more than one species of these worms may infest trout, but, unfortunately, little attention has been paid to their specific characters. From the practical standpoint, however, it is of little importance whether there is one or several species, for there is no reason to believe that they differ essentially in their habits or in their effects on the fish. They are not confined to trout, by any means, but may occur on various species of fresh-water fishes, although some of these are more susceptible than others.

DESCRIPTION OF PARASITE

Gyrodactylus may occur almost anywhere on the host but is usually most abundant on the fins, especially the dorsal and caudal fins. The affected surfaces become covered with a bluish-gray slime due to an increased secretion of mucus. Later, if the parasites are very abundant, the fins become badly frayed and may eventually be worn down to mere stubs, while open sores may appear at the base of the fins. These lesions often become infected with fungus, so that in late stages of the disease there is frequently a considerable growth of fungus on the fins and body. Gyrodactylus can be found easily by scraping off some of the slime from the affected parts and examining it under the microscope. The worms will be seen in rapid movement, twisting and squirming about in every direction.

If the fish is examined in water with hand lens, the worms can usually be seen without difficulty, attached by one end to the fish and waving the body back and forth, or they may be crawling about slowly in the same manner as a "measuring worm." Fish infested with Gyrodactylus can often be seen rubbing themselves against the sides or bottom of the pond in an evident effort to rid themselves of the worms. In fact, this is one of the most reliable indications of the presence of the parasite.

When examined under a low magnification (fig. 3) the worm appears as a small, transparent object armed at one end with a pair of large recurved hooks. Surrounding the paired hooks is a flattened,

FIGURE 3.—Photomicrograph of Gyrodactylus from trout. Magnified 85 diameters

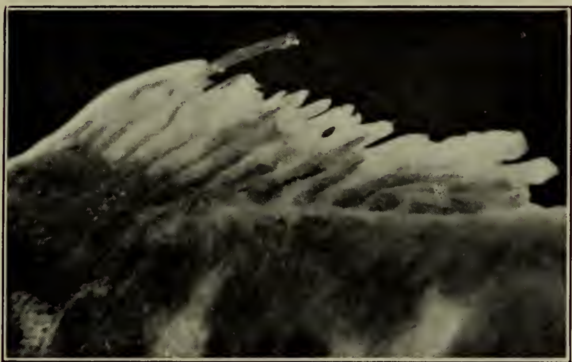

FIGURE 4.—Dorsal fin of fingerling trout infected with fin rot. Magnified 7 diameters

FIGURE 5.—Parasitic copepods (*Salmincola edwardsii*) attached to gill filaments of adult trout. Magnified 7 diameters

disk-shaped structure bearing a number of small hooks on its outer margin. It is by means of these hooks at the posterior end of the body that the worm is able to cling to the host, the hooks being embedded in the epithelium. At the anterior end the body terminates in two short lobes.

Unlike most parasitic worms, Gyrodactylus does not lay eggs. On the contrary, it gives birth to living young, which are already well developed and immediately attach themselves to the host. The young in various stages of development can usually be seen within the body of the mother, the large paired hooks being especially prominent.

CONTROL MEASURES

Although Gyrodactylus probably occurs at all trout hatcheries, for some unexplained reason many hatcheries appear to experience little trouble from the parasite while others suffer from frequent outbreaks. Fortunately, the parasite is easily controlled by dipping the fish in an acid bath. Salt solutions, which are frequently resorted to, are ineffective, although the fish may be benefited temporarily.

The late George A. Seagle, superintendent of the Wytheville (Va.) hatchery, first developed a successful treatment, in which the fish were placed in a solution composed of 1 part vinegar to 16 to 20 parts of water. This solution will kill the parasites without serious injury to the host, but since the effective agent is acetic acid, the concentration of which varies considerably in different samples of vinegar, it is better to make up a solution of acid of known strength. This treatment has been worked out carefully by Dr. G. C. Embody (1924), who used a solution containing 1 part glacial acetic acid to 500 parts water. When the fish are immersed in this solution for one minute virtually all the worms are killed without permanent injury to the fish. Doctor Embody found that an ordinary washtub about half filled with the solution was sufficient to treat about 1,000 trout 3 to 6 inches long. Since the solution rapidly becomes diluted with use after 400 to 500 fish have been treated, the rest should be allowed to remain in the solution for one and one-half to two minutes. If the fish are also affected with fungus, it is advisable, after a short time, to follow the acetic-acid treatment with a salt bath.

DISCOCOTYLE SALMONIS

Discocotyle salmonis is a trematode worm somewhat similar to Gyrodactylus but can readily be distinguished by its much larger size and dark brownish color. Unlike Gyrodactylus, it occurs only on the gills, and the worms are usually crowded in among the filaments, where they are not easily seen unless a careful examination is made. The worms, which are about 3 to 5 millimeters long, are easily visible to the naked eye. The posterior end of the worm is modified into a flattened disk, which bears on each side a row of four suckers armed with small hooks. This forms an efficient attachment organ, by means of which the parasite clings to the gills and is very difficult to dislodge.

The parasite injures the fish by sucking blood from the gills and through irritation of the tissues at the point of attachment. The gills of infested fish are usually light colored, with an excessive

secretion of mucus. When abundant the parasites may cause an acute anemia, which eventually results in the death of the host.

Nothing is known of the life history of this worm, but from analogy with related forms it is probable that the eggs, surrounded by a tough resistant shell, are laid between the gill filaments of the host, where they remain until hatched. There is no evidence that Discocotyle has an intermediate host, as is the case with internal parasitic trematodes. It is not impossible, however, that the eggs may occasionally drop from the gills and develop on the bottom of the pond. The worms apparently develop very slowly, since trout do not become infested to any extent until they are 2 years old.

This parasite was first described by Elmer Schaffer in 1916 from rainbow trout at the State hatchery, Cold Spring Harbor, N. Y. Later it was found on brook and rainbow trout at other hatcheries, but so far as the writer is aware it has been reported only from Long Island, where it is evidently firmly established.

CONTROL MEASURES

The acetic-acid treatment, which is so effective in the case of Gyrodactylus, is of no value in combating Discocotyle. The latter is much more resistant to the acid solution than the former species, and a solution strong enough to kill it usually causes the death of the host as well. Furthermore, the great majority of the worms lie between the gill filaments, where they are more or less protected from contact with any solution.

According to Laird (1927), the parasites can be successfully controlled by the use of Zonite. A solution composed of 1 part Zonite to 5 parts water is sprayed directly on the gills of infested fish by means of an atomizer. In using this treatment it is necessary to handle each fish individually, the lower jaw being bent back so that the gills are separated. If this is not done, many of the worms will not be reached by the spray.

In Europe it has been found that a closely related species occurring on the gills of trout can be destroyed by immersing the fish for one to one and one-half minutes in a saturated solution of common salt. It is claimed that the worms are virtually all killed, while the fish are not seriously injured by the short immersion in the salt solution. Apparently this treatment has not been tried in America.

PARASITIC COPEPODS

Several species of parasitic copepods occur on trout and salmon, but by far the most common form is *Salmincola edwardsii*, which is widely distributed throughout the East and Middle West. This species is the only one whose life history has been worked out, but since the other species occurring on trout are very closely related it is not probable that their behavior and life history is essentially different from that of *S. edwardsii.*

The copepods are small Crustacea, the majority being free-living forms abundant in both fresh and salt water, where they form an important item in the diet of many food and game fishes.

DESCRIPTION AND LIFE HISTORY

Salmincola edwardsii occurs only on brook trout, rainbow and brown trout being immune. The parasites are attached to the gills, where they can easily be seen with the naked eye. (Fig. 5.) They are quite large, measuring several millimeters in length, and are yellowish white in color. The anterior end of the parasite is attached to the gills by means of a special organ developed from the mouth parts. The end of this attachment organ bears a bulb-shaped enlargement, which is inserted in the gill filaments, firmly anchoring the parasite in place. Posteriorly each copepod bears a pair of long egg sacs, within which the embryos undergo complete development. The parasites ordinarily seen attached to the gills are all females. The males are much smaller and are not seen unless specially looked for.

When the young are fully developed the egg sacs break open and the larvæ escape into the water as minute, free-swimming organisms closely resembling the free-living copepods, which form such an important constituent of the plankton. They are less than a millimeter in length, very active, and swim about with rapid, darting movements. They may remain in this free-swimming stage for about two days, constantly searching for trout on which to attach themselves. If a suitable host is not found within this time, they are unable to develop further and soon perish.

Each larva possesses powerful mouth parts and a peculiar attachment filament, by means of which it is able to rasp a hole in the gill tissues, in which the enlarged end of the filament is inserted and soon becomes embedded. After attachment the parasite undergoes rapid degeneration, in which it loses its swimming feet and all evidence of segmentation, the abdomen becoming converted into a rounded, saclike structure.

About two or three weeks after attachment the parasites become sexually mature. Mating then takes place, after which the diminutive males drop off and die. The female, however, lives on for several weeks, increasing enormously in size and undergoing still further degeneration. The young are liberated in about a month after the eggs are fertilized. Each female ordinarily lays two batches of eggs, after which she dies and gradually disintegrates. Under ordinary conditions the entire life cycle is completed in about two and one-half months.

While attached to the gills the parasite injures the fish by sucking large quantities of blood and also by mechanical injuries to the tissues, which sometimes result in secondary infections with fungus. When only a few parasites are present they do comparatively little harm, but when they become very abundant, as is likely to be the case under the crowded conditions in hatchery ponds, the fish are greatly weakened and large numbers eventually succumb.

Usually adult fish are more heavily parasitized than fingerlings or yearlings, and the heaviest losses occur during the spawning season, when the vitality of the fish is low and they are consequently unable to resist the heavy drain on the system caused by the presence of the parasites.

Copepod parasites have been found frequently on wild trout in various parts of the country but are apparently rarely abundant

enough to cause serious injury. This can be readily understood, since under natural conditions it is evident that only a very small percentage of the larvæ would be able to attach themselves to the proper host. In hatchery ponds conditions are different. Here the fish are crowded closely together in a limited area with a relatively small flow of water, so that there is every opportunity for the free-swimming larvæ to find a suitable host, even though this must be accomplished within a comparatively short time.

CONTROL MEASURES

When once firmly established at a hatchery this parasite is very difficult to control. The chief difficulty lies in the fact that, like most Crustacea, the copepods are covered with a tough, resistant, chitenous membrane, which is not penetrated easily by chemicals. Consequently the parasites are uninjured by solutions that seriously affect the more delicate gill tissues to which they are firmly attached. It is therefore impossible to kill them by treating the fish with chemical solutions, as in the case of most external parasites. There is one exception to this statement that may be utilized to advantage: The larvæ are comparatively delicate and are killed in a few minutes by a strong salt solution; consequently, they can be killed while in the free-swimming stage or shortly after becoming attached, without injury to the host, but this treatment is obviously of only limited application. Owing to the large volume of water flowing through trout ponds, the cost of treating them with salt is usually prohibitive, since to be effective the treatment must be continued for some time. In the case of fingerlings frequent salt baths may be used successfully to prevent the fish from becoming parasitized, but even here it can be considered only a temporary expedient.

Fasten, to whose investigations we are indebted for most of our knowledge of this parasite, strongly recommends the installation of sand filters in all cases in which the parasites occur in the water supply. This will effectually prevent the larvæ from being carried into the ponds. Since adult trout are most heavily parasitized, Fasten also recommends that in badly infested hatcheries only 2-year-old fish be used for egg production, and that these fish be discarded immediately after spawning. Of course, this would necessitate rearing a new lot of brood fish each year, which would greatly increase the cost of the eggs.

The introduction of predacious minnows into the brood ponds has also been recommended. These fish feed on the larvæ of the parasite before they have an opportunity to become attached to a new host.

Since in most cases, at least, such methods would be only palliative and would not result in the eradication of the parasite, it would seem that in the long run it would be less expensive to get rid of all parasitized fish and start anew. Of course, in the case of hatcheries having a contaminated water supply such extreme measures would not be justified, unless at the same time an efficient sand filter is installed.

References: Fasten, 1912, 1918, 1921.

ICHTHYOPHTHIRIUS MULTIFILIIS

Ichthyophthirius is a parasitic protozoan that is quite common on pondfishes but is rarely injurious to trout. This is due to the fact that the parasite is unable to complete its life cycle where there is a rapid flow of water, so that it rarely becomes established in trout hatcheries. In a few instances, however, where the conditions were somewhat unusual this parasite has caused serious losses among fingerling trout.

The most characteristic symptom of the disease caused by this parasite, and known as ichthyophthiriasis, is the occurrence of small, grayish-white, sharply defined pimples or pustules on the body and fins. These pimples also occur on the gills but, of course, are not as readily observed as those on the body. The infected fish usually rub themselves against the sides or bottom of the pond in an effort to rid themselves of the parasite. As this is also a characteristic action of fish infested with Gyrodactylus, it should not be interpreted as a specific symptom of the disease. Heavily infested fish lose their appetite and float listlessly in the water.

The parasite can be identified easily by scraping off some of the pimples and examining the contents in water under a low magnification. They are exceptionally large for protozoans, attaining a diameter of nearly 1 millimeter, and can be distinguished with the naked eye as minute, rounded, whitish bodies swimming slowly about. Under the lens the parasites can be seen to be round or ovoid in shape and covered with an immense number of fine hairlike cilia arranged in rows, by means of which the animal is propelled through the water. At the anterior end is a small, circular mouth opening, while scattered throughout the body are numerous opaque granules and a number of small contractile vacuoles. Near the center of the body is a large, crescent-shaped nucleus.

Ichthyophthirius has a very interesting and complicated life history (Fig. 5a), a knowledge of which is essential in order to combat the disease intelligently. The young parasite is very small and quite different in appearance from the adult. It swims about actively in search of a host, and when it comes in contact with a fish it bores into the epidermis, attaching itself by one end of the club-shaped body and rotating rapidly so that it quickly displaces some of the epithelial cells. In this way the young parasite gradually works its way into the deeper layers of the epidermis, which grow over it, so that it finally lies in a closed space. If the young parasite is unable to find a fish, it dies within a few days.

Once embedded in the skin or gills of the host the parasite begins to grow rapidly and soon appears to the naked eye as the little white spot or pimple previously referred to. When full grown the parasite leaves the fish and drops to the bottom, where it soon forms a cyst by secreting a thin membrane around itself. Within the cyst it multiplies rapidly by division, and eventually a large number of minute young are produced, which are invisible to the naked eye. When reproduction is completed the cyst wall breaks open, releasing hundreds (in some instances thousands) of young, which immediately swim off in search of a new host.

CONTROL

Owing to the fact that during most of its life the parasite is embedded in the epidermis of the host, where it can not be reached by chemicals, Ichthyophthirius is more difficult to combat than most external parasites. When not embedded in the skin or gills it is easily killed by various chemicals, such as a 3 per cent salt solution or a 5 per cent solution of aluminum sulphate. In using salt, the

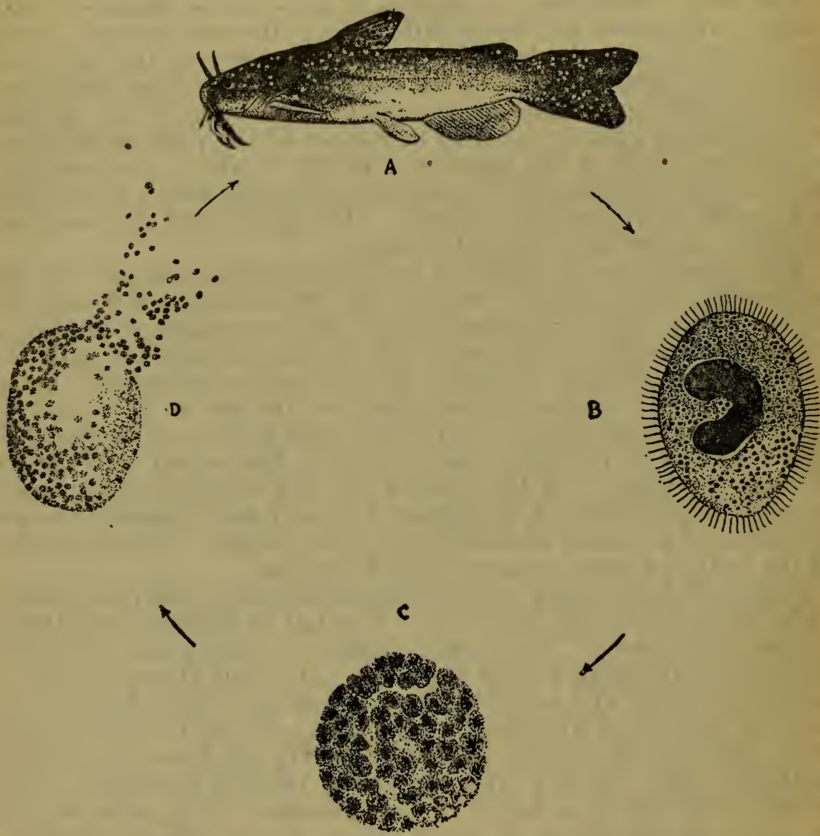

FIGURE 5a.—Life cycle of the parasite. *A*, Adult parasite on catfish; *B*, parasite after leaving fish as a free-swimming form and settling to the bottom; *C*, division of adult into many smaller individuals after formation of a cyst; *D*, bursting of cyst, releasing hundreds of minute parasites, which in turn reinfect the fish

fish may be dipped in the solution until they show signs of distress, or the salt may be added to the troughs in the usual manner. If aluminum sulphate is used, the fish should be dipped in the solution for one minute. Of course, it will be necessary to treat the pond in which the diseased fish have been held with salt or quicklime to kill any parasites that may be lying on the bottom. Since parasites embedded in the skin are not affected by the solution, it is necessary to treat the fish on several successive days in order to kill them as they emerge.

EXPLANATION OF FIGURES 6 TO 17

FIGURE 6.—Ventral view of *Chilodon cyprini*. The mouth opening leading into the pharynx is shown at the anterior (upper) end. Below the pharynx is the large nucleus, on each side of which is a small contractile vacuole. Magnified 740 diameters

FIGURE 7.—Ventral view of *Costia necatrix*, showing the two pairs of flagella. The only internal structures shown are the contractile vacuole and the smaller rounded nucleus. Magnified 1,600 diameters

FIGURE 8.—Side view of *Costia necatrix* showing attachment of flagella to wall of ventral groove. The internal structure is not shown. Magnified 1,600 diameters

FIGURE 9.—Mature cyst of *Octomitus salmonis*. Magnified 2,100 diameters

FIGURE 10.—View of Cyclochæta from the ventral side. A row of long hairlike cilia are attached to the outer margin of the ventral disk. Midway between the outer margin and the center of the disk is a complex skeletal structure composed of chitenous plates. Magnified 850 diameters

FIGURE 11.—Side view of Cyclochæta drawn on a smaller scale. Magnified 700 diameters

FIGURE 12.—Flagellate form of *Octomitus salmonis*. The deeply stained paired nuclei, surrounded by a lighter area, can be seen at the anterior end. Extending the length of the body are a pair of axostyles, to which are attached three pairs of flagella at the anterior end and a fourth pair at the posterior end. Magnified 2,100 diameters

FIGURE 13.—Epithelial cell, from the pyloric cæca of a fingerling trout containing an intracellular stage of *Octomitus salmonis*. Below the parasite can be seen the nucleus of the epithelial cell with its deeply stained chromatic network. Magnified 1,230 diameters

FIGURE 14.—Ameboid stage of *Schizamœba salmonis* from stomach of trout. Two large vesicular nuclei can be seen within the ameba. Magnified 1,640 diameters

FIGURE 15.—Cyst of *Schizamœba salmonis* showing method of division into several distinct parts, each of which contains several small deeply stained nuclei. Magnified 1,640 diameters

FIGURE 16.—Trophozoite of *Chloromyxum truttæ* from gall-bladder of trout. Magnified 800 diameters

FIGURE 17.—Mature spore of *Chloromyxum truttæ*. The pear-shaped polar capsules, each containing a small coiled filament, can be seen at one side of the spore. Magnified 2,500 diameters

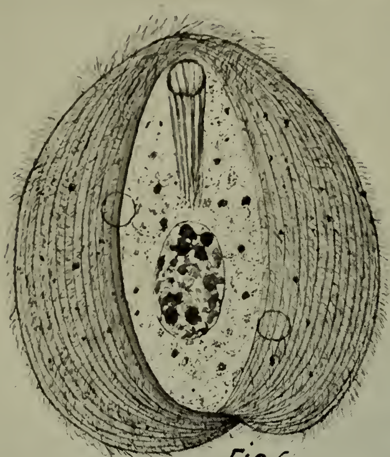

Fig. 6.

Fig. 7.

Fig. 9.

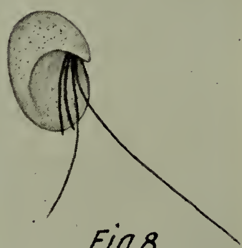

Fig. 8.

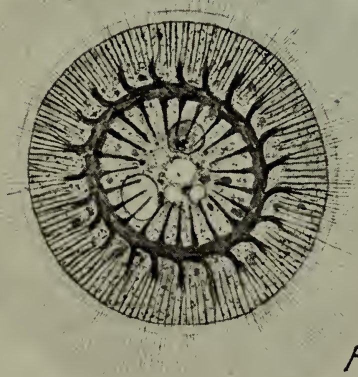

Fig. 10.

Fig. 11.

Fig. 12.

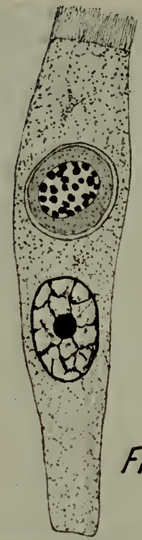

Fig. 13.

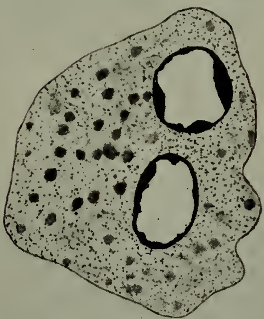

Fig. 14.

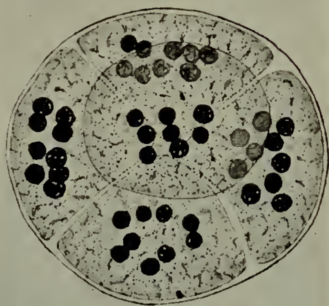

Fig. 15.

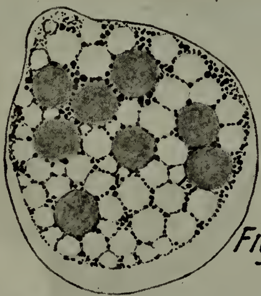

Fig. 16.

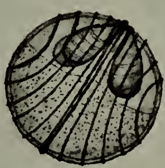

Fig. 17.

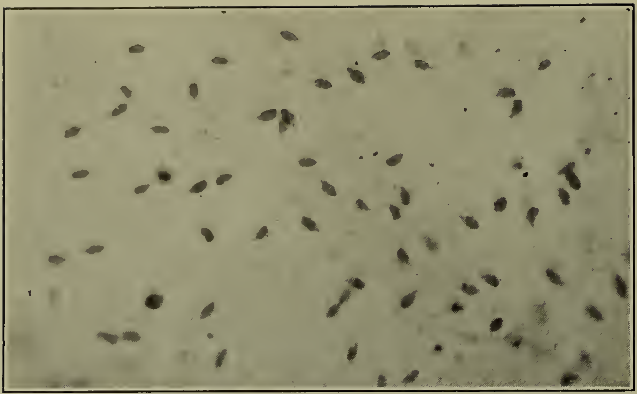

FIGURE 18.—Photomicrograph of a small drop of the intestinal contents of a trout infected with *Octomitus salmonis*. Magnified 360 diameters

FIGURE 19.—Gills of fingerling trout affected with gill disease. Note that the gill filaments are enlarged at the ends, and in many instances adjoining filaments have become fused for some distance. Magnified 14 diameters

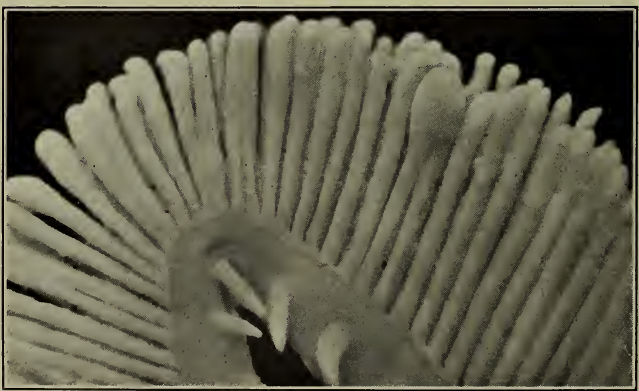

FIGURE 20.—Same as Figure 19

Owing to the difficulty of killing the parasites by means of external application of chemicals, a more practical method is to remove the adults as they leave the fish. This may be accomplished by holding the fish in swiftly running water, which carries the parasites away before they have an opportunity to multiply and reinfect the fish. This method can be adopted easily at any trout hatchery, as it is only necessary to hold the infected fish in troughs or raceways, through which a current of water is kept flowing. Of course, it will be necessary to hold the fish under these conditions for several days until all white pimples have disappeared.

Reference: Prytherch, 1924.

CYCLOCHÆTA

Cyclochæta, like Ichthyophthirius, is a ciliate protozoan. Unlike Ichthyophthirius, however, it does not burrow into the epithelium and is found only on the surface of the body, fins, and gills. Although usually present only in small numbers, it may occasionally become so abundant as to cause considerable injury to the host. It is frequently associated with Chilodon and Gyrodactylus.

Cyclochæta is different from any other parasite found on trout and can be recognized very easily. It is a small microscopic organism, discoidal in shape, with a flattened ventral side, by means of which it is attached to the surface of the fish. Under a hand lens it appears as a small, transparent, circular disk crawling rapidly about over the surface of the body and fins.

Cyclochæta has in reality a very complicated structure, which can only be made out under a comparatively high magnification. The flattened ventral surface, which is applied to the epithelium of the host, has a very remarkable chitenous skeleton composed of a series of chitenous plates forming a ring with processes extending from both the inner and outer side (fig. 10). A row of long, hairlike cilia is attached to the margin of the ventral disk, by means of which the animal moves about over the body of the host.

When the parasite is viewed from side (fig. 11) an additional row of cilia can be seen arising from a circular groove about midway between the dorsal and ventral surfaces. At one side this groove merges into a funnel-shaped mouth, which is not shown in the figure.

Cyclochæta multiplies by means of binary fission, in which the animal divides into two similar individuals of equal size. There is no evidence that it has an encysted stage, and the parasite is probably unable to live off the host for any length of time.

Cyclochæta is killed very easily; one treatment with a 3 per cent salt solution or with a 1 to 500 solution of acetic acid is ordinarily sufficient to entirely rid the fish of this parasite.

Reference: Plehn, 1924.

CHILODON CYPRINI

This is a protozoan parasite that occasionally occurs on trout and in some instances has been known to cause serious injury to fingerlings. It is a much more common parasite of pondfishes than of trout and is frequently very abundant on goldfish, causing serious mortality both

among the young and adult fish. According to Moore (1924), two distinct species of Chilodon occur on trout.

Infested fish show little evidence of the presence of the parasite until they become very abundant, when the fish lose their appetite and show a tendency to lie on one side on the bottom of the trough. When the fish are examined with a hand lens the parasites can be seen as minute, colorless, flattened organisms creeping rapidly about over the surface of the fins, body, and gills. Under a higher magnification Chilodon appears distinctly heart-shaped (fig. 6), the anterior end being somewhat pointed, while at the posterior end there is a distinct indentation. This indentation is formed by a shallow groove, which extends forward on the ventral side to the mouth opening near the anterior end. The mouth opens into a short tube, the pharynx, the walls of which are supported by a number of chitenous rods. The surface of the body is covered with short, hairlike cilia, which are much longer at the anterior end. It is by means of these cilia that the parasite moves about over the body of the host. The cilia originate from a number of concentric lines extending from the anterior to the posterior end and which give the parasite a very characteristic appearance. The animal is about 75 microns long with a maximum width of about 50 microns.

Chilodon reproduces by binary fission, each individual dividing into two by a transverse constriction. Since under favorable conditions successive divisions may occur within a few hours, the parasites frequently increase very rapidly.

While Chilodon is often abundant over the entire body of a goldfish, in the case of trout it appears to be confined largely to the gills and fins. It does not appear to injure the host to any appreciable extent unless present in large numbers, but when abundant the parasite may be the cause of serious mortality among small fingerlings.

Chilodon can be controlled easily by dipping the fish for a short time in a 3 per cent solution of common salt or in a 1 to 500 solution of acetic acid. Usually one treatment is sufficient, but in bad cases it is advisable to give a second treatment on the following day. Since the parasites readily leave the fish, especially after the death of the host, the troughs or raceways should also be given a thorough treatment with the salt solution.

References: Moore, 1924; Plehn, 1924.

COSTIA NECATRIX

Costia necatrix is a small protozoan parasite that is not uncommon on pondfishes and occasionally may become so abundant on trout as to cause serious injury. When very abundant it produces a disease known as costiasis, which may be quickly fatal.

SYMPTOMS

Probably the most characteristic symptom of the disease is the appearance of a light bluish or grayish film, which spreads over the body and fins. The fish lose their appetite, become rapidly weakened, and die in a short time. These symptoms, however, are not sufficiently distinctive to enable one to recognize the disease with certainty without a microscopical examination. This can be made easily by

scraping a small quantity of slime from the body and examining it in a drop of water under the microscope, using a comparatively high magnification. The parasites, if present, can be seen as minute oval bodies darting here and there with great rapidity.

DESCRIPTION OF PARASITE

Although very small, Costia has a complex structure, which can only be made out with considerable difficulty. The body is much flattened, with definite dorsal and ventral surfaces. When viewed from above it is oval in shape (fig. 7), with rounded anterior and posterior ends. The ventral surface is concave, with a deep oral groove extending obliquely from left to right across the body (fig. 8). This groove is much deeper on the left side, where it leads into the gullet near the anterior end. Two pairs of flagella arise from the groove where it opens into the gullet. One pair of flagella is much shorter than the other, extending only a short distance beyond the posterior margin of the body. The longer pair is two or three times the length of the body and of unequal length, one being about two-thirds the length of the other. These flagella are used for propelling the animal through the water and also for clinging to the epithelium of the host.

The parasites live on the skin and gills of the fish, where they destroy the epithelial cells, apparently feeding on the fragments. They multiply very rapidly and under favorable conditions quickly overspread the entire surface of the body and gills.

Since the parasites can leave the fish at will and swim about, for a short time, at least, it is obvious that under the crowded conditions at a hatchery there is every opportunity for them to spread rapidly from one fish to another. They may also form resistant cysts, which enable them to live for some time off the host.

CONTROL

Like all protozoan parasites that live on the surface of the body, Costia can be killed by salt baths. Since many of the parasites are embedded in the mucus, it is necessary to subject the fish to a somewhat longer exposure to the solution than in the case of Chilodon or Cyclochæta. An immersion of about 10 minutes in a 3 per cent salt solution will kill most of the parasites, but it is advisable to follow this with two or three baths at intervals of about three days to kill any that may have escaped the first treatment. The acetic-acid treatment may also be used and has the advantage that it requires only a short immersion in the solution, one minute in a 1 to 500 solution of glacial acetic acid being sufficient.

References: Moore, 1923; Plehn, 1924.

INTERNAL ANIMAL PARASITES

PARASITIC WORMS

Although quite a number of parasitic worms are known to occur in trout, they are, fortunately, very rarely so abundant in hatchery fish as to cause appreciable injury. This is in striking contrast to other types of parasites, which are almost invariably more abundant

in trout at hatcheries than in those living under more natural conditions. The answer to this seeming paradox is, no doubt, to be found in the fact that almost all endoparasitic worms require at least two distinct hosts for the completion of the life cycle. The adult worm lives in an animal known as the primary host, whereas the larva is found in a very different animal known as the secondary or intermediate host. In some cases the worm requires more than one secondary host for its complete development. The larvæ can only develop to a certain stage in the secondary host, but should this animal be eaten by the primary host they are then able to complete their development in the latter.

Trout may serve either as a primary or as a secondary host but never as both with the same species of worm. Worms for which trout form the primary host usually occur in the larval stage in some crustacean, whereas those worms that utilize trout as the secondary host usually occur in the adult stage in fish-eating birds. This being the case, it is easy to understand that there is little opportunity for these parasites to complete their life cycle in hatchery fish that, for the most part, are fed on artificial foods and protected from predacious enemies.

All types of parasitic worms occur in trout, including flukes (Trematoda), tapeworms (Cestoda), roundworms or threadworms (Nematoda), and spiny-headed worms (Acanthocephala). The flukes are so small that they are seldom noticed, although they may occur occasionally in the intestine. A larval fluke (possibly a species of Holostomum) may form cysts in the skin. Since these cysts are surrounded with pigment, they appear as minute black spots, which, when abundant, are quite conspicuous. Similar cysts are common in the skin of yellow perch, minnows, and other pondfishes.

Ward and Mueller (1926) have recently described a form of " popeye " in black-spotted trout at one of the Oregon State hatcheries, caused by heavy infestation with the larvæ of a trematode worm. The larvæ formed minute cysts in various parts of the body and were so abundant as to cause heavy mortality. The protrusion of the eyes was apparently due to the presence of cysts in the optic nerve, as only those fish in which cysts were found embedded in one or both nerves showed the popeye condition. The enormous number of cysts found in this instance can probably be accounted for by the presence of snails in the nursery ponds. From our knowledge of the life history of closely related worms it seems very probable that this parasite requires two secondary hosts. The first is a mollusk (probably a snail) in which the parasite multiplies rapidly, after which it becomes encysted in fish, which form the second intermediate host.

A number of tapeworms have been reported from trout, probably one of the best known being the species described years ago by Leidy under the name *Dibothrium cordiceps*. The larvæ of this species are very common in the muscles and body cavity of trout in Yellowstone Lake and for that reason have attracted considerable attention. The adult tapeworm is found in the pelican, which is abundant at certain seasons. Fasten (1922) has found a similar worm to be very abundant in trout from certain lakes in Washington.

Another tapeworm, *Abothrium crassum*, which is common in salmon in both this country and Europe, has recently been found in

considerable numbers at one of the Vermont State hatcheries. Only the adult worm is found in trout and salmon, where it lives in the pyloric region of the intestine.

Roundworms or nematodes are comparatively rare in trout. One species, *Cystidicola stigmatura*, has been reported frequently from salmonid fishes from the Great Lakes, and a similar parasite (probably the same species) has recently been found in brook trout from several streams in Pennsylvania. The small, white, threadlike adult worms, about 1 to 1½ inches long, live in the air bladder and are sometimes present in large numbers. The larvæ occur in the fresh-water shrimp (Gammarus).

The spiny-headed worms are characterized by a retractile proboscis, armed with numbers of recurved hooks, which is embedded in the intestinal wall, sometimes resulting in infection followed by severe inflammation. Like the tapeworms, the spiny-headed worms have no digestive tract, but unlike the former the body is small and unsegmented. One of these worms is sometimes quite abundant in European trout and is reported to have caused considerable mortality. The writer has seen specimens of brook trout from Newfoundland that were so badly infested with spiny-headed worms that they must have suffered considerable injury.

References: Fasten, 1922; Linton, 1891; Ward and Mueller, 1926.

OCTOMITUS SALMONIS

Octomitus salmonis is a small protozoan parasite that occurs in the intestine of trout and salmon. This parasite has received considerable attention during recent years because it has been shown to be the cause of serious mortality among fingerling trout in our hatcheries.

The parasite is widely distributed throughout the country, having been reported from trout hatcheries in many different localities, and it is probable that there are very few hatcheries at which it does not occur. It has not been found in wild fish except under circumstances that indicate that the infection was probably derived from hatchery fish. There is, however, no reason to doubt that the parasite does occur naturally in wild trout, but it is probably rarely sufficiently abundant to cause noticeable injury under such conditions. It is only when the fish are crowded together in the hatchery that Octomitus becomes a serious problem.

Octomitus salmonis has been found in all species of trout and salmon propagated artificially. In most cases it is more injurious to brook trout than to either rainbow or brown trout, although at a few hatcheries the rainbow appears to be more susceptible than the brook trout. It is notable that this is true only at hatcheries where rainbow trout are reared, and it is not improbable that under such conditions a physiological strain of the parasite has been developed that is more virulent for rainbow than for brook trout.

SYMPTOMS

Octomitiasis is not characterized by well-defined symptoms by means of which it can be distinguished readily from other ailments of trout. There are no external lesions, and the most common

indication of the presence of the disease is the appearance of very emaciated fish commonly referred to as "pinheads." Many of the "pinheads" may improve after a time and eventually resume their normal rate of growth, but others gradually grow weaker and weaker until death supervenes.

More rarely the disease may occur in an acute form accompanied by heavy mortality. In such cases the fish may exhibit a whirling or corkscrew motion in the water, and in some instances they have been observed to lie on the bottom of the trough and bend the body from side to side with quick, spasmodic movements.

Often a prominent feature of the disease is the "spotty" nature of the outbreak. Instead of appearing simultaneously in all the troughs containing a certain lot of fish, the disease may first break out in one or in several troughs that may not be connected in any way. It frequently happens that all the troughs containing fish from the same lot of eggs will eventually show the disease, although by the time it appears in the last of the troughs it may have run its course already in the troughs first affected.

The simplest and most reliable method of diagnosing the disease consists of a microscopical examination of the contents of the anterior end of the intestine. This material should be mounted on a slide in a drop of water, in which the parasites will remain alive and active for 10 to 15 minutes. Since no other parasites are likely to be encountered that could be confused with Octomitus, an examination with the low power of a compound microscope is usually sufficient. At this magnification the parasites can be seen easily as colorless, minute, pear-shaped organisms (fig. 18) darting rapidly about in every direction. In some instances the parasites do not occur in the cavity of the intestine but are found only in the intestinal lining. This condition, however, usually occurs only in very young fingerlings.

ETIOLOGY

As stated above, the parasite is a small protozoan belonging to the Flagellata. The members of this group are characterized by the possession of one or more long, whiplike locomotor organs known as flagella. In the case of Octomitus there are four pairs of these flagella, three of which are attached to the broader anterior end of the body while the fourth pair arises from the posterior end. In life it is very difficult to distinguish the flagella, because they are very transparent and usually in rapid motion.

Octomitus is colorless and transparent, and in order to make out the details of its structure it is necessary to kill and stain the organism (fig. 12). It is then found that there is a pair of chitenous rods, known as axostyles, extending throughout the length of the body, to which the flagella previously referred to are attached. On each side of the axostyles near the anterior end is an elongated nucleus, each nucleus being connected with the nearest axostyle.

The flagellates reproduce by a process known as binary fission, during which the organisms become rounded and the various cell structures, with the exception of the flagella, divide into two equal parts. New flagella are quickly developed, so that the daughter flagellates are identical with the mother in every respect but size.

Since the process requires but a short time, it follows that under favorable conditions the flagellates may multiply very rapidly.

At certain times cysts are formed, which can live for a considerable period outside of the host, and it is probably by this means that the parasites are ordinarily transmitted from fish to fish. The cysts (fig. 9), which are ovoidal to spherical in shape, are formed by flagellates becoming surrounded by a thin transparent membrane. Shortly after the membrane is formed the inclosed organism divides into two, and in this condition the cysts pass out of the intestine in the feces. They can remain alive in the water for days, probably for weeks, and when accidentally ingested by another fish may set up a new infection. In this they are aided by the tough, resistant membrane, which enables the cysts to withstand conditions that would quickly kill the flagellated forms.

In addition to the flagellates in the intestinal cavity, there is another stage of the life cycle (fig. 13) that is found only in the epithelial cells that line the intestine and pyloric cæca. This stage is very different from the flagellated stage previously described. It first appears as a small rounded cell, which increases rapidly in size and soon divides into a number of small cells similar to the original. These daughter cells in their turn invade other epithelial cells and repeat the cycle. Under certain conditions these intracellular stages may multiply very rapidly, so that a large percentage of the epithelial cells become infected. After a time some of the intracellular parasites develop into the flagellated form and then quickly emerge into the cavity of the intestine.

PATHOLOGY

The effects of the parasites on the host undoubtedly vary widely under different conditions, and there is still much to be learned in this regard. The evidence at hand is quite contradictory in some respects, but it is believed that much of this apparent discrepancy can be explained on the basis of the two cycles of development within the host.

It is undoubtedly true that fish may harbor large numbers of flagellates without exhibiting any noticeable ill effects. This, however, appears to be largely a matter of age and probably also of acquired immunity on the part of the host. Ordinarily, trout over 3 to 4 inches in length show little or no ill effects, even when the parasite is abundant in the intestine, while younger fish may exhibit every evidence of malnutrition under the same conditions.

Among young fingerlings the effects of a severe infestation by the flagellates are usually quite marked. The fish lose their appetite and become greatly emaciated, the large head and attenuated body suggesting the term "pinhead," by which they are commonly known among fish culturists. Such fish are usually weak and listless and in late stages of the disease may become too feeble to fight the current and are swept against the screen, where they soon expire.

This chronic form of octomitiasis is usually most prevalent during the spring and early summer, when the fingerlings are from 2 to 3 inches long. While the mortality is usually not very heavy at any time the disease may persist for several weeks, so that the total loss may be as high as 50 to 75 per cent. Chronic octomitiasis is prob-

ably prevalent to a greater or less extent at most trout hatcheries where the fingerlings are held until summer or later, although the severity of the mortality appears to be dependent on a number of environmental factors, among which unsuitable food and overcrowding appear to be especially important.

Although the chronic wasting disease just described is undoubtedly the most common result of infection by octomitus, there is another form of octomitiasis, previously referred to, which manifests itself as an acute infection accompanied by a high mortality. Such epidemics occur only sporadically and are usually not of regular recurrence year after year as in the case of the chronic form of the disease. In the majority of cases acute octomitiasis occurs early in the season shortly after the fish begin to feed, breaking out first in one trough and then in another without any apparent connection between the two.

Acute octomitiasis is caused by a rapid multiplication of the intracellular stages of the parasite, the flagellated stages frequently being entirely absent. As a result there is considerable injury to the intestinal lining, accompanied by more or less inflammation, which quickly causes the death of the fish.

While there are thus two distinct forms of the disease, it is nevertheless true that in most instances we have to deal with a combination of the two types. It is very probable that even in the chronic wasting type of octomitiasis the mortality may be due largely to the intracellular parasites, which are invariably present in greater or less numbers. In fact, a comparison of dying fish with emaciated but fairly vigorous individuals from the same lot has shown that in most instances the intracellular stages were more numerous in the former.

CONTROL MEASURES

Since *Octomitus salmonis* is so widely distributed and may occur in fish of all ages, it appears to be impracticable to eliminate the parasite from a hatchery. In fact, there is no doubt that many fish harbor small numbers of the parasite and are consequently "carriers" of the disease.

All attempts to destroy the parasites by means of drugs administered with the food have been unsuccessful, and it is believed that the control of the disease must be based on prophylactic measures. Happily, there is good reason to believe that this is entirely feasible.

All available evidence points to the conclusion that, if the fry and fingerlings can be kept in an otherwise healthy and vigorous condition, there is comparatively little danger of heavy losses from this disease; but if the vitality of the fish is lowered or they are subjected, even for a short period, to unfavorable conditions, there is great danger of a rapid increase in the abundance of the parasites, with correspondingly detrimental effects on the host.

In some respects the disease may be compared to pneumonia in man. The causative organisms are usually present in small numbers in hatchery fish and under these conditions produce no detrimental effects, but, just as in the case of pneumonia, if the vitality is lowered from any cause or the natural resistance weakened, the factors that hold the organism in check are no longer effective. Consequently,

the parasites break out of bounds and increase very rapidly, with dire results to the host.

There are many factors that may directly or indirectly cause an outbreak of octomitiasis. Among those that are most conducive to the development of Octomitus are overcrowding, unsuitable water supply, especially a deficiency of dissolved oxygen, and improper food. Owing to the inherent defects of artificial propagation it is almost impossible to avoid entirely the unfavorable effects of these factors, but certainly in most instances much can be done to improve present conditions.

It is a natural tendency to attempt to increase the output by overloading the equipment. In some cases this may succeed for a time, but sooner or later it is likely to result in disaster. The evil effects of overcrowding are, no doubt, in part due to an insufficient supply of dissolved oxygen, and this, of course, is especially noticeable when the water is not properly aerated before entering the troughs or raceways. Often the water from springs is deficient in this gas, and it should always be made to flow over an efficient aerating device to obviate any danger from this source.

A promising method of control, which has received comparatively little attention as yet, is the production of immune races of trout. There is considerable evidence that this is entirely feasible but, of course, will require rigid selection for several generations before conclusive results can be obtained. As a matter of fact, owing to the prevalence of octomitiasis there has necessarily been more or less involuntary selection in this respect at many hatcheries that rear their own brood stock. This may be a partial explanation of the well-known fact that fingerlings from hatchery eggs usually grow faster and suffer smaller losses than those hatched from eggs obtained from wild fish.

References: Davis, 1924, 1925; Moore, 1923, 1924.

SCHIZAMŒBA SALMONIS

This is one of the most common parasites of hatchery trout, but there is no evidence that it is ever seriously harmful to the host. It is usually most abundant in healthy, vigorous fish and is ordinarily not common in those that are not in good condition. This is probably due primarily to the fact that the parasite rarely occurs in numbers in fish that have been without food for any length of time.

Schizamœba salmonis lives in the stomach and intestine of all species of trout and salmon, but, like *Octomitus salmonis*, it has thus far been found only in hatchery fish or fish that might readily have become infected from hatchery fish. The amebæ (fig. 14) occur in the stomach as small, colorless organisms, more or less irregular in shape. The transparent protoplasm has a finely granular structure containing one or more vesicular bodies, the nuclei. The amebæ are found in the mucus that covers the lining of the stomach and are often very abundant. This vegetative ameboid stage disintegrates quickly when removed from the stomach and for that reason is rarely seen unless special search is made for it.

The parasite is most commonly seen in the encysted stage, which is frequently very abundant in both the stomach and intestine. In

the latter the cysts are usually found in the core of gelatinous mucoid material that forms the major part of the intestinal contents. The cysts are spherical and vary greatly in size, the average diameter being about 20 to 25 microns. They are surrounded by a thin, transparent membrane and, in addition to a number of nuclei, usually contain numerous spherical refringent bodies composed of fat. The cysts gradually increase in size and after a time divide into 4 to 11 cells of approximately equal size (fig. 15). At this stage they have a remarkable resemblance to a segmenting egg, but, of course, this similarity is only superficial.

The cysts provide a means by which the parasite may be transmitted from one fish to another. Protected by the surrounding membrane they pass out of the intestine in the feces and may live for some time in water. If eventually taken into the stomach of another trout, the life cycle is repeated.

As previously pointed out, this parasite has not been observed to injure the host appreciably, although when excessively abundant it would seem that it must have some deleterious effect, even if it is limited to the consumption of food that would otherwise be utilized by the host.

Schizamœba salmonis is much more common in fingerlings than in older fish, although in several instances the writer has found it abundant in adult trout. It is probable that most hatchery fish harbor a few of these parasites, which would explain the ease with which the fingerlings become infected.

Reference: Davis, 1924.

CHLOROMYXUM TRUTTÆ

This parasite is a protozoan belonging to a group of typically fish parasites known as the Myxospo´ridia. This group includes a large number of species found in both marine and fresh-water fishes, but trout appear to be exceptionally free from them.

Chloromyxum truttœ is found in the gall bladder of brook trout and occurs in both wild and hatchery fish. Although it is sometimes very abundant in the bile, there is no evidence that it appreciably injures the host.

The vegetative stages of the parasite, known as trophozoites, are small transparent organisms, rounded to somewhat irregular in shape (fig. 16). They move slowly about by means of a single lobe-shaped projection or pseudopodium. The pseudopodium is a temporary locomotor organ, which may be formed or retracted at will. When actively moving about the trophozoites are usually distinctly pear-shaped. Trophozoites in various stages of development are ordinarily found together, the largest reaching a diameter of 50 to 60 microns. The protoplasm of the trophozoites contains a number of large, clear vacuoles, between which are crowded numerous small, spherical granules. In the large trophozoites there can usually be seen several rounded more opaque structures, the sporoblasts, which eventually develop into the reproductive bodies or spores.

The spores (fig. 17) are spherical, about 9 to 10 microns in diameter, and are surrounded by a thin, transparent shell, which is marked by a series of concentric ridges. At one side of the spore are four pear-shaped polar capsules, two of which are slightly smaller

than the others. Within each polar capsule is a fine coiled thread, which may be extruded through a pore at the apex of the capsule under certain conditions. On the opposite side of the spore, below the capsules, is a nucleated mass of protoplasm known as the sporoplasm. When the spore germinates the sporoplasm emerges and forms a new trophozoite.

After the spores mature the trophozoite in which they were developed disintegrates and the spores pass into the intestine with the bile, eventually leaving the fish in the excrement. The spores are not uncommon in the intestines of infected fish and may occasionally be seen when search is being made for Octomitus or other intestinal parasites. The spores can live for some time in water but are unable to develop unless taken into the stomach of another fish. Once in the stomach of a trout the shell of the spore splits open, the sporoplasm emerges and makes its way to the gall bladder, thus completing the cycle.

BACTERIAL DISEASES

FURUNCULOSIS

Furunculosis derives its name from the development of "boils" or "furuncles," which is the most characteristic symptom of the disease. These "boils," however, are by no means always present, and it is then necessary to rely on other criteria to diagnose the disease. Furthermore, not all boils or ulcers are the result of furunculosis, as there is evidence that such lesions may be formed by several different organisms.

The disease affects chiefly the various species of salmonid fishes but may also occur in a large number of fresh-water and marine fishes, although it apparently occurs in epidemic form only among trout and salmon. Among the trouts, the brown and brook trout are particularly susceptible, while the rainbow trout are often considered to be immune. This is not strictly true, however, since rainbow trout may contract the disease if continually exposed to sources of infection, but under ordinary conditions there is little danger of this species becoming infected.

At first furunculosis was thought to be confined to hatcheries and fish farms, but a number of epidemics have been reported in wild fish during recent years, especially among salmon in European streams. In this country serious mortality from the disease has apparently been confined to brown and brook trout in hatcheries and rearing ponds.

In Europe furunculosis has rarely affected fingerling trout, having been confined almost entirely to yearlings and brood fish; but in America it annually takes a heavy toll among the young trout at many hatcheries.

SYMPTOMS

The symptoms of the disease are usually, though not always, well marked. As mentioned above, the most characteristic symptom is the presence of open sores on the body. These usually develop in the dorsal muscles, although they may occur on other parts of the body. Sometimes there is a marked tendency for the lesions to develop at or near the base of the dorsal or pelvic fins, and conse-

quently furunculosis has sometimes been confused with the disease known as "fin rot," which is caused by an entirely different organism.

The lesions originate from small foci, which appear to the naked eye as minute red spots in the subcutaneous tissues or among the muscle fibers. These foci contain large numbers of the causative bacteria, which increase rapidly, producing disintegration of the blood vessels and other tissues in the immediate vicinity. As a result a swelling is formed, filled with a deep red, puslike material composed of bacteria, blood, and disintegrated muscle fibers. The lesions increase in size and may eventually break through the skin, forming an open sore. In many cases, however, death may occur before the lesions break through to the exterior. This is especially true of fingerling trout, where usually the only evidence of the lesion that can be seen from the exterior is an irregular, dark blotch just beneath the skin on one side of the body. This blotch ordinarily is situated between the dorsal and pelvic fins and is probably the most characteristic symptom of the disease in young trout.

In many cases the intestine is inflamed, especially in the pyloric region, and there is often a marked congestion of the blood vessels in the abdominal cavity. The spleen is usually considerably enlarged and has a bright cherry-red color, in striking contrast to the duller red of the organ in normal fish. The kidney is also affected and may become a semifluid necrotic mass.

According to Plehn, the disease may occur rarely in a quite different form, in which the causative organism is found only in the cavity of the intestine, at least until a comparatively late stage of the disease. In such cases there are no external symptoms, and recent investigations indicate that in some instances the bacteria may live in the intestine for some time without any apparent injurious effects on the fish.

ETIOLOGY

As indicated above, the causative organism is a bacterium that occurs in the tissues in various parts of the body and especially in the blood, causing a septicæmia or blood poisoning. In other words, the disease is essentially a generalized blood infection.

The bacterium was first described by Emmerich and Weibel in 1894 as the cause of an epidemic among trout in Germany. These authors called the organism *Bacterium salmoniciaa.* Later, Marsh, who was evidently unaware of Emmerich and Weibel's work, described an organism that he had isolated from the blood of diseased trout from Northville, Mich., and which he named *Bacterium truttæ.* There can be little doubt that the bacteria described by Emmerich and Weibel and by Marsh are identical, and accordingly the specific name *salmonicida* takes precedence over the later name of *truttæ.*

The bacterium is a short rod about 2 to 3 microns long, with rounded ends, but may show considerable variation in form, especially when grown in culture media. The most important characteristic of the organism, which makes it easy to distinguish from other bacteria when growing on culture media, is the formation of a pigment that stains the media a transparent brown.

The bacteria can usually be found in the blood of infected fish, where they are present in large numbers, occurring both singly and in

large clumps. They grow best at a comparatively low temperature the optimum temperature probably being about 10° to 15° C. (50° to 60° F.). They are unable to live for any length of time at 37° C. and for that reason can not develop in a warm-blooded animal.

The manner in which the disease is transmitted has not been determined definitely, but it seems most probable that the bacteria gain entrance into the body through the walls of the digestive tract or through small wounds on the body or gills. The disease may be readily transmitted experimentally by introducing the bacteria directly into the blood or tissues. After having gained entrance to the blood stream they multiply rapidly and are carried to all parts of the body. Some of the bacteria may become localized in the muscles, where they produce the small red foci previously mentioned. These may eventually form large ulcers filled with a red puslike material containing immense numbers of bacteria. The failure of such ulcers to develop in small fish is probably due to the fish dying before the bacteria have been able to attack the muscles to any great extent. The dark blotches on the side of the body, so characteristic of the disease in fingerling trout, are caused by the infiltration of blood among the muscle fibers accompanied by some disintegration of the tissues, but death supervenes before the breaking up of the muscle fibers has become an important feature of the lesion.

Among the visceral organs, the liver, spleen, and kidneys appear to be especially liable to infection. Large masses of the bacteria can usually be found in these organs, and the tissues of the spleen and kidney may be broken down entirely.

CONTROL MEASURES

In considering methods of controlling this disease, it is well to bear in mind that furunculosis is essentially a blood disease, the bacteria developing in tissues and cavities of the body, where they can not be reached by the external application of chemicals. Consequently, it is idle to attempt to cure the fish by the use of salt baths or disinfecting solutions. Probably a certain percentage of infected fish recover from the disease, but recovery rarely occurs in fish in which the disease is so far advanced as to produce lesions.

This being the case, control measures must be designed primarily to prevent the spread of the infection to healthy fish. This is by no means a simple matter, since it has been shown recently that apparently healthy fish may act as carriers, which greatly increases the difficulty of control. In some instances the disease has run its course and apparently died out, only to suddenly break out afresh at a later time. In such cases the bacteria may have been carried over in the intestines of apparently healthy fish or possibly in the mud and débris on the bottom of the pond, and later, should the fish become weakened in any way, the bacteria are again able to get the upper hand.

The only logical method of control is to destroy all infected fish at once. These should be burned, if possible, but if this is not practicable they should at least be covered with lime and buried in pits at a sufficient distance from the ponds to prevent contamination. Dead fish should never be allowed to lie in the ponds, as putrefaction does not appear to injure the bacteria, which, when the fish decompose, are set free in enormous numbers.

All ponds in which there have been diseased fish should be isolated and, after having been carefully cleaned, should be given a thorough treatment with some efficient disinfecting solution, such as a freshly prepared solution of water-slaked lime. According to German investigators, a 1 to 100,000 solution of potassium permanganate will kill the bacteria of furunculosis and, if preferred, may be used in place of lime. In using permanganate the pond should be filled and enough of the chemical added to make a 1 to 100,000 solution. Needless to say, great care should be exercised to insure that the disinfecting solution penetrates all cracks and crevises where the bacteria may be concealed. If practicable, after having been disinfected the pond should be exposed to the sun for several days and allowed to dry out as much as possible. Of course, all implements that have come in contact with infected material should be sterilized thoroughly at the same time.

It has been claimed by some investigators that pure water is deleterious to the bacteria and that they quickly die out in water of this type, while they may live for weeks in water containing an appreciable amount of organic matter. Consequently, it has been held that pollution is a predisposing factor, but Williamson (1928) was unable to find any connection between pollution and the disease. There is some evidence, however, that warm weather and low water favor its development. Possibly the true explanation lies in the fact that pollution, high temperatures, and low water tend to lower the vitality of trout and salmon and thus make them more susceptible to infection.

Of course, every precaution should be taken to prevent the spread of the disease from one hatchery to another through shipments of infected fish. In fact, no such shipments should be allowed from hatcheries in which the disease is known to be present. Fortunately, there appears to be less danger of the disease being spread by means of eggs. There is, however, danger that the bacteria may be carried on the packing material or egg cases, and these should be destroyed or thoroughly disinfected if there is any reason to believe that they may have been contaminated.

References: Belding, 1927; Marsh, 1903; Mettam, 1915; Plehn, 1924; Williamson, 1928.

GILL DISEASE

Gill trouble may be due to several causes, but there is only one disease that is so characteristically a gill infection as to justify the term "gill disease." This disease has only recently been recognized as a distinct infection, but there is every reason to believe that it is widely distributed in this country. Probably many obscure losses among trout, which have been attributed to the water supply and similar agencies, have in reality been due to this disease.

Recent investigations have shown that the gill disease occurs in trout hatcheries in virtually all sections of the country, and it has also been found in chinook and sockeye salmon fingerlings at a hatchery on the Pacific coast. All species of trout may be affected and probably also all species of salmon, since the disease has been observed in steelhead and landlocked salmon fingerlings as well as in the chinooks and sockeyes mentioned above.

SYMPTOMS

Unfortunately, the disease is not characterized by any well-defined symptoms other than the appearance of the gills. In the early stages the fish are often somewhat weakened, but in many cases they appear to be perfectly normal until a short time before death. In the case of large fingerlings and older fish the gills are usually congested in early stages of the disease. Later the gill filaments become fused together more or less completely, while the tips become enlarged and distinctly lighter in color (figs. 19 and 20). Eventually the gills may become badly fungused, the fungus often spreading from the gills to the top and sides of the head. This fungus is purely a secondary infection and, in the case of large fingerlings and yearlings, is almost invariably present before the fish die. Infected fry and small fingerlings, however, rarely show any trace of fungus, probably due to the fact that the disease is so quickly fatal to small fish that fungus has no opportunity to develop.

Probably the most constant symptom that can be easily recognized is the greatly increased secretion of mucus by the gills. This is usually a prominent feature of the disease in fish of all ages and is particularly noticeable, as débris of various kinds becomes entangled in the mucus and may so clog the gills as to interfere with respiration. It is well to bear in mind, however, that this excessive secretion of mucus by the gills is not distinctive, since it may be due to other causes as well as gill disease.

ETIOLOGY AND PATHOLOGY

The disease is due to infection with bacteria, which forms a luxuriant growth over the surface of the gills. These bacteria occur in the form of long, threadlike filaments, which usually lie side by side to form a more or less continuous layer over the gills. These filaments, which are in reality composed of long, rod-shaped bacteria closely joined end to end, are colorless, transparent, and so difficult to distinguish that it requires very careful focusing under a high magnification to make them out as they lie closely applied to the surface of the gills. The bacteria are most abundant on the outer third of the gill filaments, where, evidently as a result of the irritation set up by their presence, there is a rapid proliferation of the epithelial cells. This results in the free ends of the filaments becoming enlarged so that they are often more or less distinctly club-shaped. One of the most striking features of the disease is a more or less complete fusion of adjoining filaments. This fusion is more noticeable near the tips of the filaments, but in extreme cases all the filaments of each gill may become converted into a continuous mass. Sometimes the gills become necrotic, but as this condition is almost invariably accompanied by fungus it is probable that this organism is chiefly responsible for the necrotic condition. The fish usually succumb in a very short time after the fungus develops.

The increased secretion of mucus, accompanied by enlargement of the gill filaments, must seriously interfere with the circulation of water over the gills. To make matters worse, the mucus usually becomes filled with sand grains and débris, which must still further impede the flow of water. The result is serious interference with

normal respiration, which probably largely accounts for the high mortality often caused by this disease among fry and small fingerlings.

Ordinarily, the bacteria are found only on the surface of the gills, but in the case of fry they may also occur on the body and fins. This may account for the great destructiveness of the disease among fish of this age. In a number of instances the disease has caused virtually a total loss among fry within a few days of its appearance.

CONTROL MEASURES

Inasmuch as the disease is caused by bacteria, which occur only on the surface of the gills and body, it is evident that it should be controlled more easily than one that is due to internal parasites. This assumption is borne out by practical experience, as it has been found that the disease yields readily to treatment with copper sulphate. On the first appearance of the disease the fish should be dipped in a 1 to 2,000 solution of copper sulphate for one minute and then quickly transferred to running water. If the treatment is carried out properly, there will be little loss unless the fish have been weakened previously by the disease. In such cases it is impossible to prevent considerable loss, as the gills have been injured so badly that many of the fish die even though the bacteria are destroyed entirely. When the disease has become established two treatments on successive days will probably be required to destroy all the bacteria; but if the fish are treated on its first appearance, one dipping is sufficient, and the loss among the treated fish is usually negligible.

It has been found that fry and fingerlings may contract the disease from older fish that appear to be perfectly healthy. Evidently the bacteria may live in small numbers on such fish for an indefinite time without producing any noticeable effects. These fish act as carriers, and it is probable that in this way the disease is carried over in a hatchery from one season to the next. It is evident that the presence of carriers will make it very difficult, if not impracticable, to completely eradicate the disease from a hatchery.

References: Davis, 1926 and 1927.

FIN ROT

This disease, which is also known as "tail rot," has recently occurred at a number of widely separated hatcheries, where it has caused considerable mortality. It has sometimes been confused with the fin disease caused by Gyrodactylus, which is entirely different, although the early symptoms may be quite similar.

SYMPTOMS

Fin rot is characterized by the disintegration of the fins, which frequently are destroyed entirely. Nevertheless, the disease is sometimes difficult to diagnose, because there may be considerable variation in the symptoms, probably due largely to differences in the age of the fish and in the virulence of the causative organism.

In small fingerlings the pectoral fins usually are affected first, the dorsal fins being attacked next, and the infection may spread even-

tually to the other fins, although they are rarely as badly affected as the pectorals and dorsals. In older fish there does not appear to be the same sequence in the order of infection, and in many instances the dorsal or caudal fins are apparently affected before the pectorals.

Ordinarily the first noticeable indication of the disease is a more or less distinct white line along the outer margin of the fin. This white streak gradually moves toward the base of the fin, while at the same time the outer margin becomes badly frayed owing to the disintegration of the tissue between the fin rays (fig. 4). This process continues until eventually the entire fin may be destroyed. In late stages of the disease sores filled with glistening white pus may develop at the base of the fins, and occasionally such lesions are found on the body at some distance from the fins. Due to the discharge of pus into the surrounding water these lesions may appear as small, usually circular depressions, which extend for some distance into the underlying muscles. The sides of such lesions are so sharply defined as to give the impression of a small piece of tissue having been removed by some sharp instrument.

At this stage the disease has been confused with furunculosis but can readily be distinguished from the latter by the character of the lesions. The characteristic sores of furunculosis always contain a red puslike material, which is very different from the white pus found in the lesions caused by fin rot. Furthermore, the development of the sores is entirely different in the two diseases. In fin rot the infection is from the outside, so that the more superficial tissues are attacked first. Consequently, the tissues are gradually eroded from the surface, forming a lesion, which looks as though it might have been eaten out by some small animal. On the contrary, in furunculosis the sores develop from small foci, which are usually deeply embedded in the muscles and gradually extend toward the surface as the bacteria invade the surrounding tissues.

The extent to which the sores develop at or near the base of the fins is largely dependent on the size and age of the fish. In young fingerlings death usually occurs before the infection extends beyond the fins, so that the occurrence of lesions on the body is rare. Larger fish, being more resistant, may live for a time after some of the fins have been entirely destroyed, and therefore there is more opportunity for lesions to develop in the adjacent tissues.

Sometimes only a portion of the fin is destroyed. This is especially true of the dorsal and caudal fins. In the caudal only a few rays may be affected, and these disintegrate, forming a deep notch in the fin, which otherwise appears to be normal. In the dorsal fin only the outer half or two-thirds may be destroyed, the basal portion remaining unaffected.

ETIOLOGY AND PATHOLOGY

The causative organism is a rod-shaped bacterium, which can usually be found in large numbers in the infected fins. The writer has found this organism in all cases of fin rot from several different localities, and apparently it is the only organism that is uniformly present in the diseased tissues.

As previously indicated, the infection usually starts on the outer margin of the fin. As a result of the irritation caused by the growth of the bacteria there is a rapid proliferation of the epithelial cells, so that the epithelium becomes greatly thickened. This thickened epithelium forms a white streak across the fin, which is so characteristic of the early stages of the disease. Later the epithelium is destroyed, exposing the fin rays, which are also attacked by the bacteria and soon become frayed and broken. On the inner side of the infected area the bacteria are continually invading uninfected tissues, which in turn pass through the same sequence of events as those infected earlier.

The bacteria develop not only on the outer surface of the fins but also invade the tissues, attacking chiefly the connective tissues. In their growth through the tissues the bacteria follow the lymph channels and frequently can be seen grouped around the fin rays in great masses. As the cartilages at the base of the fins are less resistant to bacterial action than the denser peripheral portions, they are sometimes destroyed first, and the fins may drop off instead of gradually disintegrating. Arriving at the base of the fin through the lymph channels, the bacteria proceed to invade the surrounding tissues, causing the formation of the sores previously referred to.

CONTROL

In view of the method of infection outlined above, control measures obviously must be directed toward destroying the bacteria before they penetrate the tissues. This can be accomplished by dipping the fish for one to two minutes in a 1 to 2,000 solution of copper sulphate. This treatment has been found to be effective in early stages of the disease, but after the bacteria have gained entrance to the tissues they are out of reach of chemical baths. Usually several treatments at intervals of 24 hours are required before the spread of the disease can be checked effectually. As it is impossible to cure fish in advanced stages of the disease, all such fish should be destroyed before beginning the copper sulphate treatment. If not removed, they form a continual source of infection, as great numbers of bacteria pass into the water from lesions on the fins and body. It is also essential that the troughs or ponds and all utensils used around the diseased fish be thoroughly disinfected.

The disease varies greatly in severity; in many instances only a small percentage of the fish die, while in other cases the mortality may be very high. In the milder form of the disease a large proportion of the infected fish may recover; the lesions heal before the fins are entirely destroyed, and the fins regenerate more or less completely.

Little information has been obtained regarding the factors that tend to bring on an outbreak of the disease. As in the case of so many other trout diseases, overcrowding is undoubtedly an important contributing factor. There is also evidence that Gyrodactylus may be involved in some instances. It is easy to understand how injury to the fins by this parasite might make them more susceptible to infection.

MISCELLANEOUS DISEASES, INCLUDING THOSE OF UNCERTAIN ORIGIN

FUNGUS DISEASE

Trout, like most fresh-water fish, occasionally are attacked by a fungus disease caused by the development of water molds or Saprolegniaceæ. The fungus that occurs on trout is commonly referred to as *Saprolegnia ferax*, but it is probable that several distinct species have been confused under this name.

Although the disease often causes quite heavy losses, there is every reason to believe that the fungus is not the primary cause of the trouble. Any physical injury or infection by external parasites may result in Saprolegnia obtaining a foothold on the fish, which then spreads from the original site of infection. The fungus may develop on any part of the fish and usually occurs in small definite patches but in late stages may cover an extensive area. Ordinarily it appears as a tuft of white threads, which radiate out from the body of the fish for a distance of about ⅓ inch or more. When the water is somewhat roily sediment and débris may become entangled among the fungal filaments so that the "fungused" areas appear a dirty gray or brown. The fungus is attached to the fish by means of small, rootlike filaments, which penetrate the skin and in late stages of the disease may even invade the underlying muscles. As the filaments extend through the skin, they cause the death of the surrounding tissues so that large necrotic areas are formed, which may eventually cause the death of the fish.

Saprolegnia may also attack the eggs, and at most hatcheries the heaviest losses from fungus occur at this stage. There is no evidence that Saprolegnia can begin to develop on a normal, healthy egg unless there is some foreign organic matter adhering to the surface. However, it develops very quickly on any dead eggs that may be present, and from these eggs the mycelial filaments spread rapidly to adjoining healthy eggs, which are soon killed. Consequently, within a comparatively short time a large number of eggs may become bound together in a mass by entangling filaments, which continually spread farther and farther from the original site of infection.

Saprolegnia reproduces by means of minute biciliate zoöspores, which are produced in enormous numbers in enlarged club-shaped ends of the filaments known as zoösporangia, and it is these zoöspores that enable the fungus to spread from fish to fish. In addition to a sexual reproduction by means of zoösporangia and their zoöspores, Saprolegnia reproduces sexually through the formation of egglike oöspores, which, after being fertilized, develop into mycelia similar to that formed by the zoöspores. The sexual type of reproduction occurs only rarely and is of little importance from the practical standpoint.

There is no doubt that this fungus was originally a saprophyte, developing only on dead animal matter, and that the parasite habit is a secondary acquirement. As a matter of fact, it may grow either as a saprophyte or as a parasite and often develops on dead fish or other animal matter that may be available.

CONTROL

Everything considered, probably the best treatment for fish affected with "fungus" is a strong solution of salt (sodium chloride). The

fish should be dipped in a 3 per cent solution until they begin to show signs of distress. In early stages of the disease one treatment is often sufficient, but if the fungus has become well established it will probably be necessary to dip the fish on several successive days. Dipping the fish for one minute in a 1 to 2,000 solution of copper sulphate is also effective. German fish-culturists recommend a solution of 1 to 100,000 of potassium permanganate, in which the fish are allowed to remain for 30 minutes or longer. This treatment would hardly be practicable where many fish are affected.

In dealing with "fungus," however, by far the most effective method is to prevent the development of the disease, and in most cases this is a comparatively simple matter. As previously mentioned, there is reason to belive that healthy, uninjured fish are not affected by Saprolegnia; but infection is very likely to follow any mechanical injury, even though very slight. Once the protective mucous covering of the fish is broken an opportunity is afforded for the zoöspores to germinate and penetrate the epithelium at the point of injury. From this focal point the mycelium then invades the surrounding uninjured tissues.

Infection as a result of physical injuries is especially liable to occur during or shortly after the spawning season. Not only is it almost impossible to avoid slight injuries to the fish as a result of handling incident to stripping, but, the vitality of the fish is usually lower at this time than at other seasons, so that they are especially susceptible to infection. Fortunately, the danger of spawned fish developing a fungus infection can be decreased to a marked extent by dipping them in a 3 per cent salt solution after they have been stripped.

In some cases fish that have not been handled may become infected with fungus as a result of their own spawning activities, which often produce abrasions on the fins and body. Of course, such fish should be treated at once with a strong salt solution.

Infection with external parasites such as Gyrodactylus often results in the appearance of patches of fungus on the fins and body. In fact, if the fish have not been handled recently, the widespread appearance of fungus is almost invariably an indication that the fish are infested with some animal parasite or have contracted a bacterial disease. Obviously, in such cases the treatment should be directed against the primary parasite rather than the fungus, which is a purely secondary infection.

Under ordinary circumstances Saprolegnia can be prevented from causing any considerable injury to incubating eggs by carefully removing all dead eggs at frequent intervals. If this is done, there will be little opportunity for the fungus to become established. Care should also be taken to prevent the eggs becoming covered with sediment or débris. This not only tends to smother the eggs but also affords an opportunity for the development of bacteria and protozoa, which may so injure the eggs as to make them susceptible to infection by Saprolegnia.

One of the most effective weapons in combating fungus is to keep the ponds and troughs in a sanitary condition. Any surplus food or dead fish that are allowed to remain in the ponds for any length of time become covered with a luxuriant growth of Saprolegnia, which,

of course, results in the formation of enormous numbers of zoöspores. These are continually being set free in the water, so that the slightest wound on any fish present is very liable to become infected.

The writer recently saw a very striking example of the danger of allowing Saprolegnia to grow in a hatchery. An investigation of a widespread infection by fungus among fingerling trout disclosed the fact that the fish were infested with Gyrodactylus. Although these parasites were present on every fish, they did not appear to be abundant enough to account for such a large percentage having developed fungus. On further investigation it was found that decaying food had been allowed to accumulate in the flume supplying the troughs and ponds containing the diseased fish. This food was covered with a very luxuriant growth of Saprolegnia, which was, of course, producing zoöspores in countless numbers. These spores were being carried directly to the fingerlings infested with Gyrodactylus and were evidently responsible for the exceptional growth of fungus that appeared on these fish.

Reference: Clinton, 1894.

POPEYE

"Popeye" is a popular term applied to fish that show a marked protrusion of the eyeballs. This condition is common among fingerling trout at some hatcheries and in many instances has resulted in heavy losses.

There are undoubtedly several forms of "popeye" disease, due to quite different causes. One form, caused by a severe infestation with the larvæ of a trematode worm, has already been referred to in the section on parasitic worms. Another type of "popeye," often called "gas bubble" disease, described by Marsh and Gorham (1905), is the result of the water supply being supersaturated with air. Most spring waters are more or less deficient in dissolved air, but occasionally the water may be supersaturated. In such cases there is a tendency for nitrogen gas to collect in various parts of the body, including the loose connective tissues surrounding the eyeball, causing it to protrude from the orbit. This form of "popeye" can be prevented easily by installing an efficient aerating or, in this case, deaerating device, which will allow any excess gas to escape from the water.

Still another form of "popeye" results from an accumulation of serous fluid in the abdominal cavity and other parts of the body. In this case the abdomen is greatly distended and when opened is found to be filled with a watery fluid. This is probably the most common form of "popeye" in trout and in all cases observed by the writer has been found to be associated with a diseased condition of the kidneys. The kidneys of the affected fish are usually darker in color than normal, due to the accumulation of pigment, and the tubules contain casts and crystals, probably of some calcium salt. In places the epithelium lining of the kidney tubules may be entirely disintegrated. The cause of this disease is unknown, no evidence having been found that it is due to a specific infection of any kind. Trout showing this type of "popeye" have in several instances shown a marked improvement following the addition of 1½ to 2 per cent of cod-liver oil to the diet. This suggests that the disease may possibly

be connected in some way with calcium metabolism, especially since it appears to occur only at hatcheries supplied with water having a high calcium content.

Recently the writer observed still another form of "popeye" in fingerling trout characterized, as in the previous case, by the accumulation of serous fluid in the abdominal cavity and around the eyeball. Here, however, the resemblance ends, since the kidney tubules do not contain the characteristic casts and crystals observed in previous cases but, on the contrary, show a heavy infection with a myxosporidian parasite. It has not yet been determined whether or not this parasite is the cause of the popeyed condition.

Several writers have held that certain forms of "popeye" are due to bacterial infections of the eye, but there is still considerable doubt if this is the case. In a recent study of "popeye" by Williamson (1927) the investigator isolated 22 strains of bacteria from the diseased eyes of fishes, but concluded that none of them could be proved to be responsible for the "gas bubble" disease.

References: Marsh and Gorham, 1905; Williamson, 1927.

THYROID TUMOR OR GOITER

This disease, which is characterized by enlargement of the thyroid gland, was at one time believed by some investigators to be of a cancerous nature. It is now universally conceded, however, to be analogous to goiter in man and to have no relation to cancer. The belief in the cancerous nature of thyroid enlargement in trout was largely due to the peculiar structure of the gland in fishes. Unlike the thyroid of higher animals, the gland in fishes is not surrounded by a definite capsule. On the contrary, the cells form a branching structure, which extends into the surrounding tissues. When the gland becomes enlarged it has a striking superficial resemblance to a maligant growth, since it appears to invade the surrounding tissues in a similar manner.

The thyroid is a small, ductless gland that produces an internal secretion essential to the health of the animal. It is located beneath the floor of the mouth between the first and third gill arches. Owing to its small size and the separation of its units, which are distributed among other tissues, it is not recognizable to the naked eye.

The first external indication of thyroid enlargement is a red streak or spot on the floor of the mouth near the second pair of gill arches. This red area is due to an increased blood supply to the enlarging thyroid and may appear in fish only 2 to 3 months old. This is followed by an external swelling, which may appear on the ventral side of the head just beneath the gills as a cone-shaped, reddish tumor. Sometimes the tumor first appears on the floor of the mouth and secondary growths frequently occur on the gills and at the anterior end of the lower jaw. The tumor does not become visible externally before the fish are at least 6 months old, and only rarely do they become noticeable before the fish are a year old. The greatest number of goiters are said to appear during the second and third years of life, probably due to the fact that the thyroid is most active in rapidly growing fish. In older fish the activity of the thyroid decreases, and the tumor may decrease in size and disappear,

the fish making a spontaneous recovery. Large tumors often become abraded, which results in infection by fungus or bacteria.

The primary cause of thyroid tumor is now generally conceded to be a deficiency of iodine, which is essential to the proper functioning of the thyroid gland. There is also evidence that overcrowding, overfeeding, a limited water supply, and insanitary conditions in the ponds are important contributing factors.

CONTROL

Since the causes of the disease are so well understood, its control is a comparatively simple matter. Wild trout virtually never show any evidence of goiter, and hatchery fish in early stages of the disease show an immediate improvement when liberated in natural waters.

Inasmuch as a deficiency of iodine is the principal causative factor, it is essential that this condition be corrected at once. This can be done by adding small quantities of a solution of iodine to the water, but the addition of iodine directly to the food is much simpler and apparently gives equally good results. The form in which the iodine is administered appears to make little difference in the results. At the bureau's hatcheries very good results have been obtained with the so-called "Lugol's solution," which consists of 1 per cent iodine dissolved in a 1 per cent solution of potassium iodide. A tablespoonful of this solution thoroughly mixed with about 50 pounds of ground food is sufficient to keep the fish from showing any trace of thyroid tumor.

When such products as shrimp meal, clam meal, haddock meal, or cod-liver meal, which are rich in iodine, are fed to the fish, it is, of course, unnecessary to add iodine to the ration. Even a comparatively small percentage of these iodine-rich products will serve to protect the fish from goiter.

References: Marine and Lenhart, 1910 and 1911; Marine, 1914; Marsh, 1911; Gaylord and Marsh, 1914.

WHITE-SPOT DISEASE

The white-spot disease occurs in both eggs and fry and is characterized by the appearance of an opaque or white area in some part of the embryo, usually the yolk. The opaque area is very noticeable in the semitransparent yolk, so that the disease is easily recognizable even in its early stages. White-spot may occur at any stage of development up to the complete absorption of the yolk sac but is more likely to appear during the early stages.

There is still considerable uncertainty regarding the cause of the disease, and it seems probable that it is not always due to the same agency. The characteristic white-spot in the yolk is due to coagulation of the transparent yolk, causing it to become opaque. In many instances the coagulated yolk contains one or more kinds of bacteria, but this is by no means always the case. Frequently numbers of the so-called periblast cells, which are instrumental in the absorption of the yolk, are present in the white spots.

The evidence points strongly toward the conclusion that white-spot is primarily caused by some injury to the eggs. Such an injury might produce coagulation of the yolk followed by increased activity of the

periblast cells. If the egg membrane, or in the case of the fry the layer of cells surrounding the yolk, is ruptured or perforated, any bacteria present may gain entrance and develop in the yolk, which, of course, is simply nonliving organic material.

The fact that several kinds of bacteria may occur in the white spots and that no one kind appears to predominate is strong evidence that the disease is not due to a specific infection. Furthermore, there is no indication that the disease is contagious, as is shown by the random distribution of diseased eggs among those that are perfectly normal.

It is well known among trout culturists that white-spot is most likely to occur in eggs that have been shipped some distance or that have been handled roughly. This, of course, is in complete accord with the theory that the disease is usually the result of physical injuries. There is also evidence that in some cases the disease may have been the result of the eggs having been chilled or frozen.

Reference: Leach, 1924.

BLUE-SAC DISEASE

The blue-sac disease affects the fry before the yolk sac is absorbed and usually appears within one or two weeks after hatching. The first symptom is an enlargement of the yolk sac, which soon becomes so heavy that the fish are unable to rise to the surface. The sac usually bursts after a few days, which results in the death of the fry.

The enlargement of the yolk sac is due to the accumulation of a serous fluid between the yolk and the outer wall of the sac. The fluid often has a bluish tinge, which has given rise to the name commonly applied to this disease.

The cause of the disease is not known definitely, but has been variously ascribed by different writers to rough handling of the eggs, resulting in shocks or jars, to too much pressure during stripping, and to infection by bacteria. In support of the latter view it has been found by a German investigator, L. von Betegh, that the serous fluid may contain a pure culture of a diplobacillus. He also calls attention to the fact that the disease may break out suddenly in a lot of young fry, which indicates that it is infectious. On the other hand, in many cases it has not been possible to demonstrate the presence of bacteria in the serous fluid, so that the bacterial origin of the disease is by no means proved.

As in the case of the white-spot disease, it seems probable that blue sac is usually associated with the improper care and handling of the eggs and that it rarely attacks fry hatched from healthy, uninjured eggs.

References: von Betegh, 1912; Leach, 1924.

SOFT-EGG DISEASE

The soft-egg disease is a peculiar condition of the eggs, which has caused very heavy losses at a number of commercial hatcheries in New England. The disease, which does not appear until some time after the eggs are spawned, is characterized by the eggs becoming soft and flaccid. The cause of this condition is the formation of

minute openings in the egg membrane, which allow the water to pass freely in either direction, thus destroying the turgidity characteristic of normal eggs. If these perforations are formed before the yolk sac has developed, more or less of the yolk may escape into the water, where it immediately hardens.

The openings in the egg membrane are produced by some external agency, which causes disintegration or digestion of the membrane in small localized areas. There is no doubt that the perforations are produced by some microscopic organism, but the identity of this organism has not been definitely determined. Three types of organisms usually are found in the infected areas—a fungus (Saprolegnia), bacteria of several species, and an ameba. There is little doubt that the primary cause of the disintegration of the egg membrane is either bacteria or the ameba, because the fungal filaments do not appear to be present until after the perforations have been formed. The filaments then grow through the holes and spread out on the inside of the egg membrane. The evidence at hand seems to indicate that the ameba is the primary agent rather than the bacteria, but this evidence is by no means conclusive.

Fortunately, exact knowledge of the source or nature of the causative organism is not necessary for working out efficient methods of control. Since the cause of the trouble is undoubtedly some organism that gets on the eggs after they are removed from the fish, it is evident that rigid sterilization and antiseptic methods are the prime requisites. All pans, receptacles, etc., including trays, should be sterilized thoroughly (preferably by boiling) at frequent intervals, and all troughs should be painted each season. Every precaution should be taken to avoid, as far as possible, introduction of infection from outside ponds, and in spawning and washing the eggs clean spring water should always be used. At a number of hatcheries very good results have been obtained by treating the eggs with a strong salt solution for a short time. At hatcheries where these methods of control have been adopted the loss from soft eggs has been reduced to a negligible quantity.

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